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SULFANILAMIDE IN OTITIS MEDIA IN CHILDREN. FURTHER REPORT ON A CONTROLLED SERIES.*

DR. EDWIN B. BILCHICK and DR. GEORGE HUNTER O'KANE (by invitation), New York.

In 1939 the authors studied the effects of sulfanilamide therapy on otitis media in children at the Vanderbilt Clinic and Babies Hospital in New York.[1] We reported 103 cases, which included 55 treated with sulfanilamide and 48 controls untreated with sulfanilamide during the three months of January, February and March, 1939. The results of these studies are in the process of publication and may be briefly summarized. The number of mastoidectomies was not reduced, but the average duration of aural discharge after onset of treatment was reduced by over one-half. Moderate drug dosage was employed in this series.[2, 3]

This study was again resumed during the three months of January, February and March, 1940, under the same conditions, and included 40 treated cases and 40 controls unselected in any respect; however, in view of a larger and more thorough experience with the drug, larger dosage was deemed feasible and was employed at once during this time. Where sulfanilamide did not seem sufficiently effective or was badly tolerated, sulfapyridine or sulfathiazol in adequate dosage was quickly substituted. For the ambulatory cases, which were closely supervised, with frequent visits to the clinic, the initial dosage was 200 mg. per kilo, given in divided doses in the first 24 hours, after which the amount was cut to about 120 mg. per kilo. This dosage, with bicarbonate of soda, was

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continued from four to seven days after we noted cessation of the aural discharge and all related symptoms and signs. The customary therapy was carried out in both sulfanilamide-treated and control series. Myringotomy was performed when necessary. Scrupulous care of the auditory canals, as well as adjuvant systemic treatment and care of the upper respiratory system were a rigid part of the therapy. Whenever necessary, the case was admitted to the Babies Hospital for further controlled treatment, or operation if indicated. The statistical data and results of this study are indicated in the accompanying tables.

TABLE 1. BACTERIOLOGIC FINDINGS IN 80 CASES OF ACUTE OTITIS MEDIA.

Organism	40 Cases Treated with Sulfanilamide	40 Control Cases
Beta hemolytic strept.	23	19
Pneumococcus	8	4
Type I	3	....
Type III	0	1
Type IV	1	....
Type V	1	....
Type XV	2	....
Staph. aureus	7	5
Staph. Albus	5	7
Strept. viridans	3	....
Hemophilus influenza	....	1
Micrococcus tetragenus	....	1
No growth	....	2

From the data presented, the following conclusions may be derived:

1. The duration of the aural discharge was reduced by one-third in the sulfanilamide-treated cases.

2. The number of mastoidectomies was materially reduced in the sulfanilamide-treated series (by over one-half).

3. No serious complications resulted from the sulfanilamide therapy. The four reactions noted consisted of vomiting, diarrhea and skin rashes.

4. Our results show a remarkably close parallel to the reports of some other observers, particularly the large group reported by Bowers.[4] Table 4 demonstrates the similarity to the results reported by Bowers, who also used moderate dosage at the outset of his studies and then increased the dosage in his later cases. When one compares our results in 1940, with those in 1939, although the total number of cases

TABLE 2. SUMMARY OF DATA IN 80 CASES OF ACUTE OTITIS MEDIA, INCLUDING 40 TREATED WITH SULFANILAMIDE AND 40 CONTROLS.

Number of Cases	Treated with Sulfanilamide 40	Not Treated with Sulfanilamide 40
Age	6 months to 10 yrs.	6 weeks to 9½ yrs.
Sex—Male	26	22
Female	14	18
Number with Unilateral Otitis Media	26	30
Right	13	....
Left	13	....
Number with bilateral otitis media	13	10
Total number of ears affected	52	50
Number of myringotomies	44	33
Total days of discharge after onset of treatment	325	444
Average number of days of discharge after onset of treatment	8.5	12.6
Total number of reactions	4	....
Number admitted to hospital	6	5
Number of mastoidectomies performed	2	5
Percentage of mastoidectomies	5%	12.5%
Number of simple primary mastoidectomies	2	4
Number of revisions of simple mastoidectomy	0	1
Number of complications	0	0
Number of deaths	0	0
Bacteriology of patients coming to operation:		
B. hem. strept	2	3
Hem. staph. aureus	....	1
Hemophilus influenza	....	1
Number of cases of early recurrence of discharge	5	....
Number of cases of late recurrence of ear infection	3	....

in each series is comparatively small, a definite decrease in the number of mastoidectomies (from six to two) is noted in the treated cases, while in the control series no change can be found. The average number of days of duration of aural discharge proved to be exactly the same for the two years in both treated and untreated series. This could hardly be a coincidence and is convincing proof of the similarity of the virulence of the organisms, as well as the effectiveness of the mode of therapy for the two years. The only variable to be noted was the larger dosage of sulfanilamide employed in the 1940 series, which we feel was the actual cause of the reduction in the number of mastoidectomies.

Recurrence of Infection: One of the most interesting series of observations in this group has been the occurrence of five early and three late recurrences of otitic infection after the cessation of administration of sulfanilamide and the presence of a dry ear. The time interval before the flare-

TABLE 3. COMPARISON OF RESULTS OF TREATMENT IN YEARS 1939 AND 1940.

Year	1939	1940
Average number of days of aural discharge after onset of treatment with sulfanilamide	8.6	8.5
Without sulfanilamide	12.6	12.6
Percentage of mastoidectomies performed in cases Treated with sulfanilamide	16%	5%
Treated without sulfanilamide	10%	12.5%

Note: The only variable to be noted in the two years was the larger dosage of sulfanilamide in the 1940 series.

TABLE 4. COMPARISON OF RESULTS WITH SERIES REPORTED BY BOWERS.[4]

	Bowers	Bilchick and O'Kane
Average number of days of aural discharge after onset of treatment with sulfanilamide	9	8.5
Without sulfanilamide	17	12.6
Percentage of mastoidectomies in cases Treated with sulfanilamide	5%	5%
Treated without sulfanilamide	11%	12.5%
Reduction in percentage of mastoidectomies in cases treated with sulfanilamide	50%	60%

up of the reinfection varied between one week and six weeks. In several cases repeated myringotomy was necessary, with resulting sterile culture. In view of our experience with the drug and the reports of other observers, we must conclude that we are not dealing with a new infection but with a flare-up of a latent process which had not been completely controlled by the sulfanilamide. These recurrences were not found in the control series. The explanation of this phenomenon undoubtedly lies in the failure of the body to react immunologically to the invading organisms. The production of natural antibodies may very well be inhibited by the drug. This opinion has been expressed by Converse,[5] who investigated the antibody content of the blood in three cases of meningitis which followed mastoidectomy and sulfanilamide therapy. The antibody content was found to be low in these cases. Furthermore, animal experiments have shown that sulfanilamide-treated animal survivors of hemolytic streptococcus infection fail to acquire protective antibodies as a result of their experience. These considerations indicate the necessity for further development of adjuvant treatment of the infections with serum antibodies, bacterial immunogens and transfusions from immunized donors. Concentration of these problems may bring us to the heights of therapeutic prowess. Sulfanilamide should not be considered the final mode of therapy in otitis media and mastoiditis.

Because of alteration of the clinical picture by sulfanilamide, increased vigilance is necessary in order to recognize and treat surgically those cases in which surgical mastoiditis with bony necrosis is masked by the drug. Numerous cases of this sort have been reported and, unfortunately, have served to act as a deterrent to the use of the drug in suitable cases. As a corollary, we urge caution in resorting to hasty surgery of the mastoid where toxic symptoms such as high fever due to chemotherapy simulate the onset of intracranial complications. At the advent of such symptoms the drug should be discontinued. When the patient's true status has been determined, the proper therapy may then be instituted.

Summary: Our studies in sulfanilamide therapy in otitis media in children have shown that administration of the drug results in reduction in the duration of the discharge by one-third and a reduction in the number of mastoidectomies. No serious complications resulted from the drug in fairly large dosage.

Comment: We urge the thorough treatment of all severe cases of acute otitis media with sulfanilamide or its allied compounds, using large dosage, careful observation, and hospitalization when needed; the drug should be continued for five to seven days after the cessation of all symptoms; the necessary surgery, including myringotomy and mastoidectomy should not be postponed or omitted when indicated; reliance should not be placed upon the drug alone; immunological therapy should be employed. With the employment of these modes of therapy the outlook for rapid recovery in acute otitis media has been greatly improved.

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876 Park Avenue.

116 East 53rd Street.

AUTOPSY STUDIES OF A CASE OF TUBERCULOUS PERIPHARYNGEAL ABSCESS.*†

DR. SAMUEL IGLAUER, Cincinnati.

From within outward the pharynx consists of three principal layers or tunics; namely, a mucous membrane lining, a middle muscular layer, and an external connective tissue covering, or adventitia. The latter is of especial interest in the study of peripharyngeal abscess, as will be described later on.

External to the pharynx there are several anatomic spaces containing loose areolar tissue. Laterally lie the paired peripharyngeal or pharyngomaxillary spaces through which course the nerve-vessel bundles. Behind the pharynx there is an extensive single, wide space, the retropharyngeal space, continuous below with the space behind the esophagus, the two together constituting the retrovisceral or postvisceral space. All of these spaces are external to the outer coat of the pharynx and, therefore, may be designated as extramural spaces, which physiologically permit free movement of the pharynx during rotation of the neck and during the act of deglutition.

Peripharyngeal Space: In contradistinction to these extramural spaces, two intramural spaces contained within the connective tissue coat of the pharynx have been described by Weintraub.[1] These paired spaces, which this author designates as a right and a left peripharyngeal space, are separated in the median plane by a vertical fibrous septum. Laterally, each space extends to the nerve-vessel bundle on its own side. Superiorly, the space terminates in a blind pouch or cupola on a level with the upper margin of the superior constrictor muscle; and inferiorly, the space terminates behind the cricoid cartilage (see Figs. 1 and 2).

The peripharyngeal spaces as such have not been described by other authors but Grodinsky and Holyoke[2,3] describe two spaces external to the pharynx (between the pharyngeal and

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†From the Department of Otolaryngology, College of Medicine, University of Cincinnati.

the "alar" fascia and designated as spaces No. 3), which have approximately the same vertical and mesial extent as the peripharyngeal spaces but which extend into the neck laterally and anteriorly beyond the nerve-vessel bundle.

Pathology: It is generally conceded that an abscess external to the musculature of the pharynx, originally designated

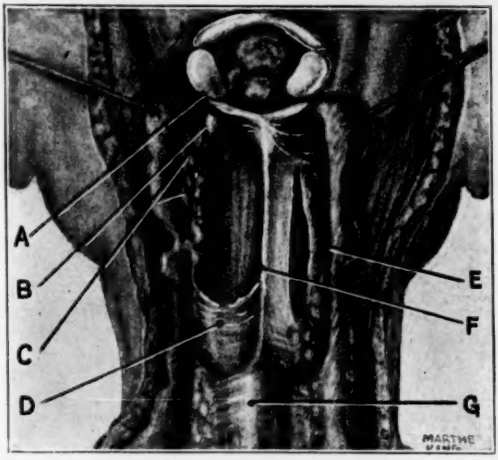

Fig. 1. Newborn infant — neck viewed from behind. (A) The atlas; (B) lateral retropharyngeal lymph gland; (C) chain of fat; (D) left peripharyngeal space laid open; (E) right peripharyngeal space partially opened; (F) fibrous septum between the peripharyngeal spaces; (G) esophagus.

as an idiopathic retropharyngeal abscess, originates from the disintegration and rupture of a suppurating lateral retropharyngeal lymph gland.[4,5,6] This gland (often called Gillette's[7] gland) is adherent to the outer coat of the pharynx and lies in the interval between the cupola of the peripharyngeal space and the internal carotid artery (see Figs. 1 and 2).

Until quite recently it has been the general belief that pus discharging from such a suppurating gland would enter the retropharyngeal space and give rise to an abscess within this space. On the other hand, as I have pointed out in some detail elsewhere,[8] Gillette's gland (or glands) lie in intimate topographical relation to the upper outer wall of the peripharyngeal space; and pus discharging from the gland, fol-

lowing the path of least resistance, would tend to enter and distend the peripharyngeal space rather than the retropharyngeal space, with which it is not in such intimate topographical relation (see Figs. 1 and 2). (As pointed out by Weintraub,[1] the cupola of the retropharyngeal space lies at a lower level, and the peripharyngeal space intervenes between the gland and the retropharyngeal space.)

Furthermore, the clinical site of the abscess and the anatomic site of the space coincide. Since both the abscess and the space lie posterolateral to the pharynx musculature and

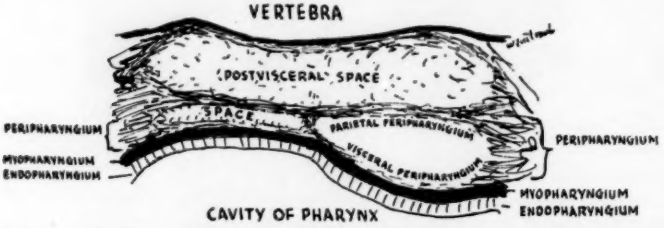

Fig. 2. Diagram showing the relationship between the layers of the pharynx, the mural paired peripharyngeal spaces and the unpaired postvisceral space. On the observer's left the normal relationship is shown; on the observer's right the peripharyngeal space is shown as though distended, with an abscess bulging the pharyngeal layers forward toward the cavity or lumen of the pharynx. Note the endopharyngium, the innermost mucous membrane layer; the myopharyngium, the middle muscular layer; and the peripharyngium, the outermost adventitial connective tissue layer. The peripharyngium consists of the visceral peripharyngium applied to the myopharyngium, the peripharyngeal spaces, right and left (one shown distended), separated by the median peripharyngeal septum, and the parietal peripharyngium. Behind the peripharyngium and in front of the vertebral column with its ligaments is the unpaired postvisceral space (Weintraub[1]).

each terminates at the midline, the lower limits of the space and of the well developed abscess also coincide, and this would not be the case if the pus were free in the retropharyngeal space, from which it could easily descend into the retroesophageal space and give rise to mediastinitis. (Clinical experience has shown that this complication is of very infrequent occurrence.)

The ordinary acute abscess is usually due to infection of the retropharyngeal lymph gland with common pyogenic organisms.[9,10] On rare occasions the gland may be invaded by the tubercle bacillus. Numerous authors[4,5,6] have described autopsy findings in such cases in which glands were found in various stages of infiltration, caseation and necrosis often

associated with abscess formation external to the pharynx. The abscesses in tuberculous infection develop at a comparatively slow rate, and the clinical course is also more gradual and less distressing to the patient. (These abscesses must not be confused with the cold abscess associated with caries of the cervical vertebrae, in which the pus collects behind the connective tissue coverings of the spine.)

Owing to the low grade inflammatory process and the slow development, tuberculous lymphadenitis in the retropharyngeal region offers an excellent opportunity for both clinical and pathological study, as demonstrated in the following case of peripharyngeal abscess.

CASE HISTORY.

B. F. S., age 44 years, a veteran of World War I, was admitted to the Hamilton County Tuberculosis Sanitarium, July 20, 1940.

Past History: His history dated back to December, 1924, at which time a positive diagnosis of pulmonary tuberculosis had been made. Subsequently he was sent to five veterans' hospitals in succession. He left the last hospital in June, 1940.

On Admission: The admission diagnosis was chronic fibroid pulmonary tuberculosis, bilateral and far advanced. This diagnosis was confirmed by positive X-ray chest findings and by the presence of tubercle bacilli in the sputum. At the time of admission he showed no evidence of tuberculosis elsewhere in the body.

During his stay in the sanitarium he was given therapeutic pneumothorax, right (July 24, 1940). Three months later he had developed hydropneumothorax, and four months later tuberculous empyema became manifest. On Dec. 27, 1940, cervical adenitis was noticed on the left side.

On Feb. 13, 1940, his throat was examined by Dr. H. G. Nelson. Despite the fact that the patient had made no especial complaints concerning his throat, a protruding swelling about the size of a hen's egg, occupying the left half of the pharynx and extending from the nasopharynx to below the level of the epiglottis was noted. The mass was fluctuant and

had rather thin walls. Posterior to the upper portion of the sternocleido muscle on the same side there was also a bulging, fluctuant area. Pressure at this point seemed to indicate that the swelling in the pharynx and the one behind the muscle communicated. There was no evidence of caries of the cervical spine, and subsequent Roentgenograms showed no abnormalities of the spine.

A diagnosis of tuberculous peripharyngeal abscess was made.

Feb. 18, 1941: A long aspirating needle was inserted behind the sternomastoid muscle, and about three ounces of yellowish, thin pus was obtained. After the pus had been aspirated, the swelling in the pharynx immediately disappeared.

Feb. 23, 1941: The patient suddenly went into collapse and expired in a few hours, apparently as a result of a spontaneous pneumothorax.

Autopsy: Feb. 23, 1941: Autopsy revealed the presence of far advanced pulmonary tuberculosis with tuberculous empyema, right, with additional (tuberculous) lesions in the bowel and prostate.

The tongue, pharynx, larynx and upper portion of the esophagus were removed *en bloc*. During their removal, an abscess ruptured. On the posterior wall of the left half of the pharynx there was a large ovoid cavity with slightly thickened walls, 2 to 3 mm. in thickness (see Fig. 3). The cavity was lined by cheesy, yellowish material and presented a slightly granular appearance. It extended from the upper border of the pharynx to the beginning of the esophagus and was definitely limited medially by a vertical fibrous septum. The left internal carotid artery extended along the outer margin of the cavity. Beneath the midportion of the lateral wall of the cavity there was a medium-sized lymph node. The long axis of the cavity measured about 7 cm., and the greatest width about 3.5 cm. Its capacity was estimated to have been about 10 cc. (see Fig. 3).

Some time later the right (normal) peripharyngeal space was opened and was found to terminate medially at the fibrous septum. (There was no evidence of caries of the cervical vertebrae.)

Microscopic Report: "Several sections show abundant muscular tissue. Along one surface there are areas of ulceration and granulomatous inflammatory reaction. These areas apparently represent the wall of the abscess noted in the gross examination. Relatively little necrosis is present, but there is distinct organization and a moderately abundant giant cell reaction that is suggestive of granulomatous inflammation.

"Another section shows in addition a portion of a recognizable lymph node. Here also there is fairly typical granu-

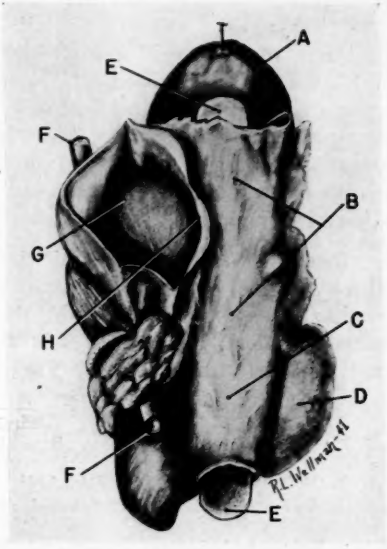

Fig. 3. Pharynx and upper part of esophagus viewed from behind, showing abscess cavity. (A) Indicates the tongue; (B) the posterior wall of the pharynx; (C) the esophagus; (D) the thyroid gland; (E-E) a tongue blade in the pharynx and esophagus; (F-F) the internal carotid artery; (G) the abscess cavity; and (H) the median fibrous septum.

lomatous inflammatory reaction with necrosis, round cell infiltration, epitheloid cell reaction and Langhan's giant cell formation. The picture here is strongly suggestive of acid-fast infection."

Microscopic Diagnosis: "Peripharyngeal abscess (probably tuberculous) granulomatous inflammation involving lymphoid tissue and regional soft tissue."

Laboratory Findings: Smears and cultures of the aspirated pus were negative for bacteria. Three inoculated guinea pigs developed positive involvement with tubercle bacilli.

COMMENT.

This case presented an unusual opportunity for the study of the pathological anatomy and the localization of a peripharyngeal abscess. The abscess cavity on the left side corresponded in its outline extent, boundaries and topographical relations to a distended and infected left peripharyngeal space.

In this particular case it is evident that the abscess occupied the left peripharyngeal space and that it had no direct connection with the retropharyngeal space, which is commonly supposed to be the seat of such abscesses. Furthermore, the findings help to explain certain clinical features of such intramural abscesses; namely, their posterolateral position, their strict limitation at the midline, and their failure under ordinary conditions to descend into the posterior mediastinum or into the adjacent peripharyngeal space.

It would, therefore, seem advisable to limit the term "retropharyngeal" abscess to designate purulent collections occupying the retropharyngeal space. It would also seem more accurate to restore the term "peripharyngeal" abscess (*abcés péripharyngiens*), formerly used by many French writers.

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707 Race Street.

SURGICAL ASPECTS OF CARCINOMA OF THE LARYNX.*

DR. JOHN D. KERNAN, New York.

Up to 10 years ago it was considered by surgeons that the only possible treatment for cancer of the larynx was surgical. A leading surgeon in a paper written about that time stated that radium or X-ray had no place in the treatment of cancer of the larynx. Several who discussed that paper expressed the same opinion. It must be said that up to that time the results with X-ray or radium had not been favorable. Great improvement in radiotherapy has been made in the last 10 years. For my own part, I present the possibilities of surgery.

Intrinsic Growths: Cancers of the larynx are classified as intrinsic and extrinsic. Intrinsic cancers are located on the vocal cords, in the ventricles or the interarytenoid space. At least 85 per cent are on the vocal cords. The epithelium of the cords is keratinizing, and tumors here are of low malignancy. Moreover, since lymphatics are scant, they metastasize late.

Extrinsic Growths: Extrinsic tumors are located on the epiglottis, ventricular bands, aryepiglottic folds, arytenoids and the pyriform sinuses. The epithelium of these regions is nonkeratinizing, and tumors here are as a rule undifferentiated. Moreover, since the lymphatic supply is free they metastasize early. Intrinsic tumors and extrinsic tumors must be treated differently.

To the most surgical of schools, so to speak, belong those who believe in total laryngectomy for every case of cancer of the larynx, however small the growth and wherever situated. Some even go further than this. A number of surgeons insist that when the larynx is removed, at the same time a complete dissection of the neck should be done on each side in order that no possibility of recurrence in the lymphatics

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may be left. These extreme radical notions are held by a minority. Numbers of competent men maintain that for many cases of cancer of the larynx a local removal suffices. I plan to deal with the various surgical procedures in the order of their severity.

Procedures done for diagnosis: *1.* Direct Laryngoscopy. *2.* Biopsy.

Direct Laryngoscopy: The operation selected depends on a number of facts, as, for instance, the size of the growth, the situation and the microscopic structure. The size and the situation are determined by mirror examination or, if necessary, by direct laryngeal examination. This is an extremely useful procedure in many cases. For you will remember that anterior commissure growths, those in the depth of a ventricle or subcordal space are exceedingly hard to visualize with the mirror. Direct examination does away with this difficulty.

Biopsy: At one time it was thought that the taking of a biopsy was an exceedingly risky procedure because it was supposed that any such trauma to a malignant neoplasm led to its speedy spread through the lymphatics. The probability is that there is no real foundation for such an opinion and I think it can be safely said that a biopsy should be taken on every laryngeal growth, even though it is already known to be malignant, in order that its histological structure be ascertained. The present author prefers to perform a biopsy through the direct speculum because he believes that this offers the best opportunity for examination and securing of an appropriate piece of tissue. The probability is that this procedure should always be carried out in the hospital with trained assistants. In a few cases that have been reported, it has been possible to totally remove a carcinoma of the vocal cord when doing a biopsy. The author has had one such experience in the case of a middle-aged man in whom a biopsy showed early carcinoma of the vocal cord. A laryngofissure was subsequently done and the cord removed. Examination of the cord showed no disease. I would suggest that no one depend on such a fortunate occurrence when treating a carcinoma of the larynx.

Methods of surgical removal: *1.* Suspension Laryngoscopy. *2.* Laryngofissure and Cordectomy. *3.* Hemilaryngectomy.

4. Total Laryngectomy. *5.* Total Laryngectomy with Neck Dissection. In judging the efficiency of any of these measures we must consider to what extent they fulfill the basic principles of cancer surgery; that is, remove the primary growth and possible lymphatic extensions.

Suspension Laryngoscopy: A few attempts have been made to dissect away small carcinomas of the cord under suspension laryngoscopy. I believe some favorable results were secured but I have heard of no one recently using this method. One would not expect that this exposure would be sufficient for dealing with such a serious disease, even though the base of the growth is destroyed by surgical diathermy. Of course, this method does not control the lymphatic fields at all.

Laryngofissure and Cordectomy: Many years ago when American authors were insisting on total laryngectomy for all cases of carcinoma of the larynx, Sir St. Clair Thomson in England was advocating cordectomy through the laryngofissure exposure. He gave indications as follows: The growth should be small, limited to one cord, and should not have involved either the anterior or posterior commissure. He considered that limitation of motion indicated involvement of the posterior commissure. Thomson by no means always stuck to his limitations. Many of the cases which he reported had much more extensive growths than those he describes and yet his favorable results were about 75 per cent of the cases reported upon. Of course, this method is very unsatisfactory for the 25 per cent not cured. Twenty-five out of 100 is a high mortality.

Jackson at the present time in this country advocates what he describes as the 1 cm. rule; that is to say, if an incision can be made all around the growth, 1 cm. from it, it is safe to remove it by the laryngofissure approach. He even deals with anterior commissure growths by this method, removing portions of both cords if necessary. Variatians of the technique of this operation are described by various authors, which I will not go into extensively here. Some prefer to remove the thyroid cartilage on the involved side, some leave the cartilage; some leave in the tracheotomy tube for 24 hours and also a feeding tube. Some close up the whole incision at once. It is said that in well selected cases, as many as 90 per cent, five-year cures may be expected.

As to the postoperative course, as a rule the tracheotomy tube if left in can be removed in 24 hours and the patient can be allowed to swallow water within a few hours. If all goes well the patient will be out of bed in a day or two and out of the hospital in a week. The healing of the interior of the larynx is slow, taking a month or two. Granulations may at times be very disturbing. When healing is complete, there will be a useful though rather inflexible voice. For dealing with early small carcinoma of the larynx situated on the vocal cords, there is no doubt that cordectomy is by far the best surgical procedure. When the growth is larger, if it has invaded, for instance, the ventricular band or the subcordal region or the arytenoid region, the mere cordectomy should not be considered. Some more extensive procedure is necessary to remove all the growth and cure the patient. When the growth reaches these regions it must be remembered that lymphatics are liable to be involved. The vocal cords themselves, as you know, are scantily supplied with lymphatics. Growths limited to them rarely metastasize. Other parts of the larynx are very well supplied with lymphatics and when the surgeon deals with growths so extensive it is always necessary for him to have in mind the possibilities of lymphatic extension.

In deciding whether or not to do a cordectomy, the mobility of the cord should be especially noted. Thomson pointed out that when the cord was perfectly mobile, he got 84 per cent cures. When the mobility of the cord was interfered with, the percentage of cures fell greatly, so that if the arytenoids were fixed, the cures were only 44 per cent. Another element to be considered in selecting an operation for cancer of the larynx is the histological picture. Local removal is quite sufficient for the well differentiated tumors. On the other hand, a highly malignant tumor, however small, calls for wide removal. It has been demonstrated that the recurrences of Grade 1 carcinomas are between 6 and 7 per cent, whereas carcinomas of Grade 2 recur in 72 per cent of the cases.

Hemilaryngectomy: Hemilaryngectomy is done with the idea of preserving an air passage which will permit the closure of the tracheotomy wound, and of preserving a useful voice. I think it is agreed among surgeons that a hemilaryn-

gectomy is more difficult to do and is attended by a higher mortality than even a total laryngectomy. It is pretty well accepted that a larynx so extensively involved as to call for a hemilaryngectomy should have a total laryngectomy done. The mortality will be less and the result about as satisfactory.

Another operation that has been suggested for the more extensive growths is the so-called Harmer operation. This involves removing one side of the thyroid cartilage or making a window in one side of the thyroid cartilage and so exposing the base of the neoplasm. Radon seeds or needles are then to be inserted in such a way as to attack directly the base of the growth. Another procedure that is used to expose a neoplasm for attack by radium is a laryngofissure incision. I feel that these methods do not belong to the surgical aspects of carcinoma of the larynx and merely mention them in passing, turning them over for fuller discussion to the radiotherapists.

Total Laryngectomy: At one time total laryngectomy was accompanied by a more forbidding mortality. Even when the patient recovered, it was liable to be after extensive sloughing in the neck, and at times a bronchopneumonia. Technique has been so greatly improved that at the present time some authors have reported series of cases with the mortality of 2 or 3 per cent. The healing time has been greatly reduced and total healing will at times be accomplished in three weeks. Frequently the tracheotomy opening is so successfully handled that it will not be necessary for the patient to wear a tracheotomy tube. With instruction, nearly every patient will be able to learn to talk. So that in discussing a total laryngectomy with any patient, unless the case is extremely advanced, one is justified in saying that he will recover from the operation, will probably not need to wear a tracheotomy tube and will have a useful voice. This is by no means so gloomy a picture as that frequently painted of the postoperative results of laryngectomy.

Indications for Laryngectomy: If an intrinsic growth has reached either the anterior or posterior commissure, the subglottic space or the ventricular bands, a total laryngectomy should be done. No form of local removal will suffice. Moreover, if the growth has invaded the ventricular band, or the aryepiglottic fold, and if the biopsy shows it to be of a highly

malignant nature, serious consideration must be given to the lymphatic fields. In my discussion on laryngectomy I will not lay great emphasis on the varying technical details but rather on the surgical necessities that must be met.

Crowe, of Baltimore, a short time ago published a paper on laryngectomy, in which he described an operation designed to save every possible tissue and to promote healing in the shortest possible time. He uses a vertical incision in the midline of the neck, preserves all the prelaryngeal muscles and performs the closest possible dissection all about the larynx. His immediate results are extremely favorable. Healing in his cases takes place almost by primary intention and his cases are up and out of the hospital in a surprisingly short time. If one, however, investigates his cases it will be seen that some of them were rather small early growths, even such as would be treated by cordectomy in the hands of other surgeons. I think it is to be questioned whether quite such a close removal of the larynx is all together safe, providing a total laryngectomy is called for.

The incision which appears to be most favorably regarded in this country is the T-shaped incision. The horizontal bar of the "T" following the line of the hyoid bone, and the vertical bar being in the midline of the neck. The triangular flaps thus marked out are well nourished and will unite with a minimum of sloughing. Innumerable incisions have been suggested, as for instance, the vertical incision uniting two horizontal incisions, one at the top and one at the lower end of the neck, or the tongue-shaped incision with its base up along the hyoid bone, or its base down along the top of the sternum. This wider incision permits examination of the regions along the great vessels of the neck on either side, and removal of the lymphatic nodes if such procedure appears to be called for. Whatever incision is called for, it should be planned so as to furnish well nourished flaps, as there will always be some danger of infection, and unless well nourished it may slough.

Thus far, in discussing laryngectomy it has been assumed that a one-stage operation is to be done. At times, however, when the growth is large, the patient will enter the hospital suffering from dyspnea. Under such circumstances it is not safe to proceed at once with a total laryngectomy. A pre-

liminary tracheotomy should be done to relieve the dyspnea and restore the patient's strength. When the reaction of the tracheotomy is over, then the laryngectomy may safely be proceeded with.

There are certain advantages in this two-stage procedure even though it is not forced on one. When such a tracheotomy is done, adhesions form in the lower part of the neck, which close the fascial planes leading to the mediastinum, and to a great extent do away with the danger of descending infection. There is, moreover, considerably less danger from bronchopneumonia when the preliminary tracheotomy is done. The disadvantage is that the adhesions make the dissecting and freeing of the trachea and larynx more difficult. Some authors, having in mind the advantage of the protection adhesions give, strongly advocate the two-stage operation. As, for instance, Gordon New proposes that a preliminary tracheotomy should be done and the larynx freed from all its connection except those with the pharynx. After the reaction from the tracheotomy is over, he allows the neck to become infected by tracheal secretion. When the reaction of all this infection has subsided, then he removes the larynx, closes the pharynx and secures quick union.

A startling proposal in connection with laryngectomy has been made by Portmann, of Bordeaux, France. It is his contention that it is impossible to prevent infection of the neck when doing a laryngectomy and, consequently, that there will always be danger of sepsis and bronchopneumonia.

He proposes, then, a two-stage operation; first, a tracheotomy, then following with a laryngectomy. He doesn't, however, close the opening into the pharynx but sews the skin edges to the edges of the mucous membrane about the opening into the pharynx, leaving a large opening into the pharynx. In this way he completely seals off the neck from infection. At an appropriate time later, two or three weeks, he closes the pharyngeal opening by plastic operation.

We have used this procedure a number of times at the Presbyterian Hospital and found it to be very successful. It is possible to close the pharyngeal opening and have it unite by first intention within three weeks after the laryngectomy, and that is a quick healing. This procedure appears

to me to be so important that I have asked Dr. George R. Brighton, who has investigated it more thoroughly than any of us, to discuss it later during the general discussion.

Total Laryngectomy with Dissection of the Neck: It has been said that intrinsic tumors do not invade the lymphatics early. Nevertheless, if the growth is advanced enough to call for a laryngectomy, the possibility of lymphatic invasion exists; therefore, when doing a laryngectomy one should always expose the regions along the vascular sheath on each side, and if any nodes are enlarged the whole lymphatic chain should be removed. This is in accordance with a firmly founded principle of cancer surgery. It will probably at once occur to you that a growth which has invaded the lymphatics is no longer intrinsic, which will be true. Any neoplasm, whatever its site of origin, or whatever its size, if it has reached the ventricular bands, aryepiglottic folds, epiglottic or arytenoids, even though grossly it appears intrinsic, is theoretically already extrinsic, and the surgeon must be prepared to deal with lymphatic extensions; so, laryngectomy with dissection of the neck is the proper operation for all extrinsic cancers of the larynx.

Another possibility of treatment exists: that is a combination of surgery and radiotherapy. Extensive growths of the larynx require such heavy doses of X-ray for a cure that the cartilage of the larynx will also be destroyed. It is proposed that laryngectomy first be done, succeeded by X-ray therapy for the control of possible lymphatic extension. A reversal of this plan is also suggested: that is, a preliminary course of radiotherapy. reducing the infection if any is present in the larynx, and destroying microscopic outlying invasion; then it is thought that laryngectomy may more safely be proceeded with. I will leave these procedures to be discussed more thoroughly by the radiotherapists.

I have purposely left out in my discussion of this subject all mention of radiotherapy. I have had Dr. Lenz treat for me a number of cases of early carcinoma of the vocal cords and some more advanced cases. The results of the early cases have been brilliant. I recall to mind one or two in whom all trace of disease had disappeared, and the final results have been a perfect larynx. I do not know, of course, how permanent the results will be. The more advanced cases

which have been treated by radiotherapy, which I have observed, have not done so well. There is always severe reaction in the cartilaginous structures of the larynx, there is liable to be perichondritis, and if a tracheotomy is required, the result is often a speedy death. Some of those in whom the growth is cured lose nearly all the larynx from sloughing and have to wear a tracheotomy tube anyway. The impression is, they would have been better off with a laryngectomy.

142 Cases Reported: 32 laryngectomies; eight five-year cures; five three-year cures; eight one-year cures; four lived less than one year but survived operation; seven died.

11 Laryngofissures: Two died; two had recurrence and laryngectomy performed; one of these died after the laryngectomy, and the other is living after three and one-half years. The other seven are living and well.

26 Tracheotomies: 15 tracheotomies after X-rays; 11 plain tracheotomies; 22 deaths; one Harmer operation; three had radon seeds — died.

103 East 78th Street.

THE OFFICE TREATMENT OF VERTIGO (HISTAMINE THERAPY).*

DR. JOHN J. SHEA, Memphis.

Vertigo is the chief complaint of one-tenth of the patients being treated for chronic conditions in our offices. They are seldom specific in describing their vertigo and frequently confuse it under the terms of headache and foolish sensations in the head. When the triad of deafness, tinnitus and vertigo are present, the condition is known as Ménière's disease. It will not be the purpose of this paper to enter into a differential diagnosis of the various types of vertigo but to present an empirical form of treatment which will be successful in the majority of cases.

The internal ears and their intracranial pathways must be disturbed for us to have vertigo. Being nerves of special sense, they are susceptible to toxins and more vulnerable to a selective group. When one compares the susceptibility of the nerves of the eye and those of the ear to the poisonous effects of methyl and ethyl alcohol, they will find a counterpart in the toxins accumulated in the body. The ear mechanism is readily influenced by toxins produced by the liver, gall bladder and colon in contrast to those of the kidney upon the optic nerves. Dwyer,[1] in 1925, nominated histamine as the culprit of common colds and suggested that their prevention be accomplished by the elimination of the absorption of histamine in the colon. The present-day treatment of vertigo is a histamine therapy.

HISTAMINE.

Histamine (β-iminoazolylethylamine $CH(NH):C(N.CH).CH_2CH_2NH_2$) was isolated by Barger and Dale in 1910, while investigating the active principles of ergot. It was found to occur naturally in many plants and animal sources (ergot, soy bean, muscle, intestinal mucosa and white blood cells). It is capable of stimulating the physiological response of

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most tissues. Administered orally, it is destroyed by an enzyme histaminase and must be given subcutaneously or intravenously. It will lower the blood pressure by increasing the permeability of the capillaries and their dilatation. It will increase the pressure of the cerebrospinal fluid by its action upon the vessels of the meninges and brain. Epinephrine is the specific antagonist of histamine.

THERAPY.

Horton[2,3] appreciated the possibilities of histamine for the relief of vertigo while using it in the treatment of erythromelalgia of the head. Today, many clinicians throughout the country are administering histamine because it is safe and effectual. The method of administration depends upon the severity of the symptoms. If the vertigo, with accompanying nausea and vomiting, is acute and severe, the drug should be given as an intravenous injection, being repeated daily until the symptoms have subsided. In my cases none have exceeded three days. If the symptoms are mild, the administration may be subcutaneous and given in the manner of a desensitizing agent.

Test: A 0.5 cc. of histamine acid phosphate (2.75 mgm. in 5 cc.) is administered hypodermatically and the patient is asked to observe if the symptoms produced are those of which he is complaining. When the response is positive, a better prognosis may be given for the therapy.

Intravenous Method: The patient should be hospitalized. One milligram of histamine base (2.75 mgm. of histamine diphosphate) is dissolved in 250 cc. of normal physiologic salt solution and administered at the rate of a drop per second. The initial symptoms will be the result of the dilatation of the vessels of the face, to be followed by those of the intracranial contents. Some of the patients become nervous and require being assured of their safety. An ampoule of epinephrine with a syringe is kept accessible in case any untoward major symptoms arise. For safety's sake, it is best to record the blood pressure before the administration, and each succeeding 15 minutes. If the stomach is empty, there will be less nausea, though occasionally the patient appreciates the powerful stimulating action of the drug on gastric secretions. Many of the nauseated patients are capable of eating the next meal, and it is preferred that they do so.

Subcutaneous Method: A 5 cc. solution of 2.75 mgm. of histamine diphosphate is preferred, especially when desensitizing the patient, and for this purpose the initial dose of 0.5 cc. will contain the equivalent of 0.1 mgm. The dose may be increased or maintained at this level two or three times a week for the prevention of future attacks. It is better to err by unnecessarily continuing the therapy than stopping short of relief. The patient should be instructed to return immediately if a relapse of the symptoms occurs.

Contraindications: Headache and dizziness are both symptoms frequently caused by intracranial pathology. It is, therefore, desirable to eliminate the various types of tumor, aneurysm, hemorrhage, hypertension, arteriosclerosis, etc., before using histamine as a test.

If the desensitization is done in doses small enough not to result in any appreciable reaction, no harm should result if one of these other conditions is present.

Cholagogue: Inasmuch as the toxins derived from the liver, gall bladder and colon have an affinity for the ear mechanism, a valuable supporting therapy should be administered to eliminate these toxins. A modification of Hammond's mixture is given once or twice daily and usually continued after the histamine therapy.

CONCLUSION.

Vertigo is a frequent complaint of patients encountered in office practice, and its relief by histamine therapy is appreciated by these sufferers.

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1018 Madison Avenue.

FRACTURES OF THE MAXILLARY BONES.*

DR. DOUGLAS B. PARKER, New York.

The great demand for speed today in all forms of locomotion and transportation has materially increased the type of accidents involving the face and head. This speed subjects the occupants of automobiles and airplanes, boats and trains to hazards not previously encountered.

In this great area of traumatic injuries, the head is exposed to a large proportion of such injuries. This is readily understood when we realize that most of these accidents happen while the persons involved are in a sitting position and the sudden impact of the vehicle causes them to be thrown forward, frequently bending from the waist to throw the head forward as the first point of impact with a stationary body. Usually the face receives the blow if the distance of travel is short. If it is longer, the skull may get the first direct blow. In automobiles, the face is liable to hit the steering wheel or the instrument panel or windshield. If the passenger is in the rear seat, the back of the front seat may be the obstacle of impact. As fast as hazards increase, safety measures are being devised to cut down the incidence of accident, or minimize the injury. In the automobile we find sponge rubber being used on the backs of seats, knobs and levers on instrument panels are being sunk or depressed below the surface. In airplanes, open cockpits are being padded and safety belts are employed in takeoffs and landings.

In industry, fibre helmets are being used in hazardous occupations, and in sports specially devised headpieces and masks are employed to prevent injuries to the face and head. Despite these various precautions, however, a great many fractures of the bones of the face continue to occur. Fights still account for many injuries, either from direct blows from a fist or missile, or from the victim being knocked down or falling against an immovable object. Blackjacks or clubs in the hands of unlawful individuals may account for a small

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percentage of maxillary fractures. Sports of various kinds, from tennis to horseback riding, may also contribute their share. Of course, we are all familiar with the fact that the war in Europe is accountable for a great many facial injuries. The recipients of these injuries are not only the military personnel but the civilian population who are caught as innocent bystanders.

Fractures of the superior maxilla occur from trauma exerted, in the main, in one of three directions: *1.* a direct blow from in front, in a horizontal plane; *2.* from above at a downward angle; or *3.* from a lateral force. Those from directly in front may involve the teeth, alveolus, maxilla or malar bones, as well as the mandible. Those from above almost always involve the nasal bones, as well as the mandible, and those from the lateral aspect generally include the zygoma or temporal bones, as well as the maxilla or mandible.

The most frequent types of maxillary fractures are those involving the alveolar plates and the teeth. The teeth may be fractured, or an entire segment of alveolar process, including several teeth *en bloc,* may be separated from the body of the bone. A fracture from a descending blow will usually fracture the nose and cause a fracture of the maxilla horizontally through the maxillary sinuses. Such a fracture is of great interest to all rhinologists because of the invasion of these sinuses. Hemorrhages into the sinuses, hematoma or ecchymosis is generally characteristic of such injuries.

In fractures of the maxilla, abnormal mobility may be one of the symptoms. This can be elicited by manual manipulation. The absence of such mobility does not imply that a fracture does not exist. Hemorrhage may or may not be a symptom. Bleeding from the mouth is usually a sign that the fracture is compound. Bleeding from the nose or pharynx usually follows many fractures where the line of fracture extends through the maxillary sinuses. Because of the character of the anatomical structures involved in the bones of the face, an impacted fracture often occurs, with resultant depression of the contours of the face without any free movement of the parts.

The Roentgen ray must be freely employed in the diagnosis of maxillary fractures. Many pictures taken in the routine

way may not delineate the lines of fractures well, and special technique will often have to be applied. Stereoscopic plates are often necessary to diagnose these conditions to best advantage because of the superimposition of so much bony structure in the ordinary flat projections on film.

The treatment of fractures of the maxilla depends on the amount of deformity, the position and number of lines of fracture, and the involvement of surrounding tissues. The same conditions hold true of fractures about the face as in fractures in general. The earlier the parts can be put back into their normal relationship and fixation maintained, the better the healing. It is always well to manipulate and disturb the primary blood clot as little as possible. In these facial fractures it is always well to have the nose and accessory sinuses checked by a rhinologist, as he understands these areas much better than the maxillofacial surgeon. Where the skull has been fractured, it is always wise to have a neurological surgeon see the patient in consultation.

If the fracture involves the alveolar process, it is extremely important to get early fixation, because there is a certain degree of terminal circulation of the parts, which, if allowed too much movement and dislodging of primary clots, will readily become infected and the bone and teeth will be lost. Displaced parts can generally be manipulated manually into their position, and if sufficient teeth are in place, and especially if posterior teeth are firm, these can be used as anchorages. By using a labial arch of either round or half-round wire of fairly heavy gauge, the teeth can be wired to this arch and can be firmly splinted together. A cast or molded splint can be used for fixation, but this implies taking an impression of displaced parts and the necessary laboratory work which delays the time of instituting fixation. This lost time may represent the difference between success or failure to get the most satisfactory results.

A fracture of the body of the superior maxilla may be unilateral or bilateral. If the fracture only involves the one side, the opposite side may act as a fixed support for anchorage if there are sufficient teeth in place to use for attachments. In such cases an arch splint can be used, or a molded splint, especially if it is light in weight. Where the fracture is bilateral, and especially if there is any downward displace-

ment, upward traction must be used to bring the parts upward into normal position and still keep the occlusion relation of the teeth in relationship, that the patient will have a useful functional result with a good cosmetic alignment of the teeth. In these complete fractures, our method of treatment is guided somewhat by the condition of the teeth. The simpler method of dealing with such a fracture is by using the mandible, where sufficient teeth are in place to occlude, to force the maxilla into position. This is accomplished by using upward traction by a chin strap or chin cup, exerted by the use of elastics attached between the chin strap and a fixed head appliance. Such a head appliance can be made by utilizing crossed straps of two-inch material over the top of the head and attached to a circular band about the head, circling just above the ears and well down over the occiput. Some men have utilized a plaster of Paris head cap with hooked attachments for the traction elastics. This is a makeshift appliance at the best, and is not very comfortable for the patient. A very simple appliance can be made by attaching safety pins about two inches apart on the side of a Barton bandage and fastening elastics between the two safety pins on each side.

I have modified this by making a chin strap of two-inch adhesive plaster with a half-inch cardboard strip held in place by folding the adhesive over the cardboard and putting the safety pins through the cardboard. This is done so that the elastics do not bunch up the adhesive tape where the elastics go over the safety pins. Another set of adhesive straps goes over the top of the head and crosses and comes down the side of the head in front of the ear. Cardboard and safety pins are used here and placed with a two-inch gap so that the upward traction of elastics can be used in connection with the pins. This method is applicable without special apparatus which has to be prepared in a laboratory or workshop.

Another method of producing upward traction is by the use of a Kingsley splint, which utilizes a cast or molded splint on each side, which protrudes from the mouth and curves to fit the contour of the face. Two traction straps attached to these side-arms pull the appliance upward by attachment to a head appliance. Such a splint as described has to be carefully adjusted so that the upward traction keeps the occlusal plane of the maxillary teeth in the same parallel plane as the

lower, so that the biting function will allow the perfect interlocking or occlusion of the upper and lower teeth when the appliance is removed. If the fixation of maxillary fractures is undertaken soon after the injury has occurred, the parts will go back into position easier and the length of time of fixation is shortened. As there is practically no muscle attachment that tends to displace the bones of the maxilla, the early fibrosis tends to hold the parts in position. Usually the fixation is quite firm after three weeks. In a large percentage of fractures of the maxilla there is some degree of hemorrhage into the maxillary sinuses with the formation of a hematoma. Fortunately, most of these clots absorb within a reasonable time and infection of the sinus is not often a sequela. It is usually necessary to use adrenalin or ephedrine in the nares in the form of drops, topical applications, or by a spray. Keeping down intranasal congestion and promoting normal drainage from the sinuses avoids many of the infections that ordinarily might follow such complicating fractures.

Impacted and depressed fractures of the bones of the face often result from traumatic facial injuries. The zygoma and malar bones, as well as the nasal bones, are the ones most frequently involved. The treatment of the fractures themselves is not a problem, because the parts tend to heal in the resultant positions following the trauma. The treatment of this type of fracture is for cosmetic reasons. The depression of the bones results in a deformity by a change in facial contour. In order to restore this contour, it may be necessary to introduce a blunt hooked instrument through a small soft tissue incision. A strong blunt or padded instrument can be used in the mouth to manipulate some of the malar fractures. A small incision in the temporal region and the introduction of a strong blunt elevator downward under the zygomatic arch may, by manipulation and force, replace the depressed fractures of the zygoma. Where the body of the superior maxilla is badly depressed, it can be handled by opening the antral wall by a Caldwell-Luc incision intraorally and elevating the bone by a blunt instrument.

Fracture of the nasal bones, which occurs so frequently with maxillary fractures, can be replaced by intranasal manipulation if seen early. Nasal splints or rubber tubing

can be used, as well as intranasal packs, to maintain the bones in position. An external moldable splint of sheet lead covered with dental modeling compound can be used in connection with adhesive straps to augment, from the external aspect, the intranasal treatment.

A great deal of mechanical ingenuity may have to be used in the handling of fractures of the bones of the face. A trained maxillofacial surgeon is the best one to cope with these conditions. They frequently require the co-operative efforts of the rhinologist and the maxillofacial surgeon who can work together to best advantage. The Army is establishing maxillofacial teams to function in all the general hospital units in event of our participation in a war. These will be in addition to mobile maxillofacial teams who will work in the evacuation areas.

There is nothing that does more to disturb the psychology of an injured patient than facial deformities which he may have to carry with him through his contacts in life. This condition is accentuated when it occurs in the female. It is for this reason that we are training a group of specialists in maxillofacial surgery, and we recognize that the greatest co-operation that we can expect is from the otolaryngologist.

HEMANGIOMA OF THE ANTRONASAL SINUS SUCCESSFULLY TREATED BY RADIUM.*

DR. SAMUEL MORSE, New York.

Mrs. J. C., age 55 years, presented herself for examination on Jan. 27, 1939. Since September, 1938, she had had frequent severe and alarming nosebleeds from the right side. They were always spontaneous, without any provoking cause. The nose was always stuffy and at times there was a good deal of purulent discharge from the right side. The patient had lost considerable weight and was anemic, worried and weak. She had never received any help or benefit from treatment, and no definite diagnosis had been made as to the cause of her trouble.

Past History: The patient had tuberculosis 23 years ago, for which she was treated and cured. She now has only a calcified lesion of the left lung, and all examinations are in every other way negative.

She had antrum sinusitis on the right side many years ago. In all probability an intranasal opening was made, judging from the appearance of the nose. It appears that the bleeding comes from this opening.

When bleeding of the nose began in September, 1938, the patient consulted a physician, who performed electrocoagulation on the right side where the bleeding tissue was seen. This was not successful, and on Sept. 20 she was admitted to the French Hospital, where a resection of the tip of the right turbinate was done for biopsy. No clinical or other diagnosis was made, although malignancy was ruled out. It was thought that there was an inflammatory hyperplasia or a benign tumor. No mention was made of the vascularity of this tissue. After this minor operation, the patient suffered severe pain and high temperature while in the hospital. She was discharged on Sept. 28, 1938, free of pain.

On one occasion this patient consulted me for excessive lacrimation in the right eye. She gave a history of having

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had the right lacrimal sac removed, following which the accessory lacrimal gland was excised.

The bleeding recurred shortly thereafter, and on many occasions until she was seen at my office on Jan. 27, 1939.

Any or several of the following diagnoses were considered:

1. Right pansinusitis (suppurative) with granuloma or polypi.

2. Synechia of the middle turbinate (right side).

3. Small septal ulcers (anterior), not large enough to cause the severe hemorrhages which occurred.

4. Opening in the lateral nasal wall, at the floor of the nose, below the inferior turbinate (two inches back from the tip of the nose). On probing and inspection of the nasopharynx, it appeared that the bleeding came from this opening. Examination with the nasopharyngoscope revealed tissue in the opening which looked like organized blood clots or granulation tissue. On later examination it had more the appearance of vascular tissue.

X-rays taken on March 17, 1939, showed a definite obliteration in the right ethmoid area and a markedly deviated septum; the antra showed no definite shadows, and the frontal and ethmoid sinuses were clear.

Some of the treatments instituted were as follows:

On Feb. 7, 1939, the synechia on the right side of the nose was incised and packed. This did not control the bleeding. On Feb. 16, galvanocauterization of the ulcers on the right side of the septum was performed. The patient was given injections of liver extract because of the low hemoglobin (65 per cent).

Certain food allergy tests were performed and an elimination diet prescribed. The latter seemed to help. The bleeding was controlled from time to time but every once in a while the patient suffered severe attacks which incapacitated her for a day or more. Vitamins were given, with calcium lactate internally, and still there was but slight relief from the bleeding, which varied from slight oozing to severe hemorrhage.

On May 29, 1939, the patient was sent to the New York Hospital; Ear, Nose and Throat Clinic for diagnosis. The

report was that the apparent cause was postoperative adhesions, right nasal cleft. Lysis of adhesions and possible turbinectomy was advised. The Wassermann test, etc., was negative.

This diagnosis did not seem to bear out the experience I had had with this patient. She had no adhesions in the nose, although there was more or less gluing of the nasal tissues due to discharge and blood clots.

The patient was exceedingly careful, but even when sitting perfectly quiet she could develop a severe bleeding attack. She had a complete medical examination at this time, which showed nothing unusual. The blood pressure was 120/80.

On July 26, 1939, while at home, the patient developed an attack of severe bleeding from the right side of the nose. She was rushed to the Brooklyn Eye and Ear Hospital, where the nose was packed. This did not control the bleeding, so a postnasal sponge was applied and she was transferred to the hospital ward. On July 28 the packing was removed from the right nostril, at which time no bleeding was seen. The following day the patient complained of severe pain over the right antrum, and of pain in both ears. She was given a spray for the nose and discharged on July 31. No diagnosis was made, either on admission or discharge.

After this occurrence the patient consulted me again and it was decided that something must be done. I was considering the question of radium for hemangioma. On Aug. 30, 1939, on examining the nose with the nasopharyngoscope when the patient was not bleeding, there was seen a small mass of tissue on the floor of the nose (right side) and also some tissue from the ethmoid area. This appeared to be very vascular and did not appear granulomatous. It was thought that radium would be an excellent form of therapy if the diagnosis was correct. The case was discussed with the radium therapist, who concurred in this opinion. At this time the patient was referred to another rhinologist, who suggested a radical operation on the antrum. I did not feel that this was feasible or practical; in fact, I felt it would be dangerous.

With a diagnosis of possible hemangioma, the patient was referred to Dr. Wallace Morrison for further consultation.

His report was: "Angioma of the paranasal sinuses. Radium therapy would be the proper method of attack."

On Aug. 31, 1939, 25 mg. of radium element in 1 mm. brass filter was applied intranasally for 1½ hours (37.5 mg. hrs.). The same amount at a distance of 2 cm. from the bone was applied externally over the right antrum for the same length of time. This was followed by another sitting of 1¾ hours (44.0 mg. hrs.) on Sept. 6, 1939. From the date of the first radium application the bleeding was controlled, and under no condition of examination or probing was there any further bleeding. On Sept. 11 the same dose of radium was given, internally and externally. A slight redness over the cheek was the only reaction, and this disappeared in a few weeks following application of ordinary vaseline.

The appearance of the nose changed. The tissue could no longer be seen. There was a slight recurrence of bleeding on Sept. 21 and radium was again applied.

At no time had it been possible to get a piece of tissue without stirring up a lot of bleeding. On April 6, 1940, the patient came in for re-examination of the nose, and a piece of tissue was obtained for examination. The report of the pathologist was as follows:

"*Gross:* Specimen consists of a small sliver of grayish-white tissue removed from the antrum for biopsy study. *Microscopic:* The tissue is composed of numerous dilated, edematous alveoli lined by cuboidal epithelial cells and resting in a scanty fibrotic stroma. Around the periphery are seen isolated areas of polynuclear infiltrations and remnants of mucosal lining. Some of the alveoli show epithelial hyperplasia. The entire structure shows evidence of radiation therapy. No malignant changes are seen. *Diagnosis:* Adenomatous polyp."

COMMENT.

In looking over the history of this case, it seems that clinically no other kind of growth could cause such profuse bleeding without provocation except an angioma. An angioma might appear histologically to be an adenomatous polyp after radiation, and since the patient had a great deal of radiation therapy it is probable that the tissues shriveled and atro-

phied. It is unfortunate that we were never able to obtain tissue for biopsy during the time when the patient had the recurrent attacks of bleeding. The biopsy taken at the French Hospital was from the turbinate. Our own biopsy was taken very much later, after radiation therapy, and this tissue examination was really done to rule out possible malignancy.

At no time did the patient have any enlargement of the glands nor did she look cachectic. She was very anemic after these attacks; and as soon as she recovered somewhat and her hemoglobin level became higher, she would start to bleed again.

In communicating with Dr. Morrison in regard to this case, he said it was a very rare condition and that he and Dr. Hurd had seen but one other such case. I have seen many cases of severe nasal bleeding but in every one the condition could be arrested by some simple form of surgery, hemostatic technique or internal therapy. Sometimes the elimination of a systemic disease like syphilis would do it. In this case everything else was ruled out and the only possible diagnosis left was hemangioma. Dr. Morrison's interest and kindness in assisting and supporting our diagnosis gave moral support to the necessity of using radium in this case.

It is important to recognize and reiterate the fact that radium and high-voltage X-ray irradiation can be used in many nonmalignant conditions. The literature is full of such reports. If there is sincere co-operation between the otolaryngologist and the radium therapist, successful results can be obtained, as I think this case indicates.

This patient had a chronic suppurative right pansinusitis which would occasionally flare up, cause considerable discharge, both nasal and postnasal.

It was our intention to operate for this condition at some future date (when the danger of spontaneous bleeding, or postoperative bleeding from the tumor mass, had been removed).

336 West End Avenue.

TWO UNUSUAL SYMPTOMS OF EYE-STRAIN.

DR. JOHN R. NOYES, Brockton, Mass.

After one has been working and thinking along certain lines for several years, he usually, if observant, learns many things that are not referred to in textbooks. This is especially true of the practice of medicine. A review of the literature has failed to reveal any mention of two symptoms which, I have found, are often due to eye-strain; namely, globus hystericus and tinnitus aurium.

Several years ago, a woman in middle age consulted me for the relief of a very annoying tinnitus and deafness, from which she had suffered for several years. She said that she could stand the deafness but that the constant head noises were driving her "nearly insane." Examination showed a well advanced case of so-called dry catarrh of the middle ear. The time-worn methods of treatment, including removal of the tonsils, cauterization of the lower turbinates, inflation and massage, were tried but little progress was made.

On one of her visits the patient complained of trouble with her eyes and asked to have them examined and new glasses prescribed. Examination revealed a marked uncorrected hyperopia, together with some astigmatism and presbyopia, all of which were no doubt causing a serious drain upon the nervous system. About 10 days after the change in glasses the patient came for further ear treatment and stated that the head noises had disappeared.

Since the change in glasses was the only deviation from the line of treatment that this woman had been given for some time, it occurred to me that there might be some connection between the tinnitus and eye-strain. To prove the point, she consented to go without her glasses for a few days. She reported that the ringing in the ears returned three days later, but disappeared a few days after the glasses were resumed. The patient soon abandoned treatment of her ears because no improvement could be made in her hearing. About

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two years later, she asked for a re-examination of her eyes because the ringing in the ears had returned. A few days after the change in lenses, she reported that the tinnitus had stopped. I was by this time fully convinced that the eye-strain was the cause of the tinnitus.

Since this experience I have been constantly on the lookout for similar ones and have been able to relieve many patients of this annoying symptom. It is sometimes present when no demonstrable ear defect can be found. Naturally, the relief of eye-strain will not effect a cure in all cases of tinnitus, but if the possibility of its presence is carefully considered in each case, many patients can be relieved by the correction of refractive errors and muscular imbalance.

Globus hystericus is another symptom of eye-strain which is not commonly considered as such. Numerous cases of this condition have come to my attention. Formerly, I was able to do very little for these patients, but continued study convinced me that the symptom was due to some disturbance of the nervous system and not to any abnormality in the throat, since many persons who complained of it had perfectly normal throats. Believing that this was a reflex nerve condition, and knowing that eye-strain is a fruitful source of such symptoms, I began working along these lines, and was gratified to learn that a large percentage of the patients were relieved by properly fitted glasses. The following case furnishes a striking illustration.

Mrs. B. was referred by her physician on account of a throat trouble which had been present for several months. Her chief complaint was a sensation as of a lump in her throat and as of someone gripping the throat with both hands, causing constriction. Dyspnea was very marked at times. She would awake suddenly at night and gasp for breath. While being examined she had a very severe attack of spasm of the glottis, became cyanotic and presented an alarming picture.

Examination of the throat showed a rather pale and atrophic mucous membrane. The tonsils were small, with no evidence of disease. The lingual tonsil was not enlarged and the nasopharynx and larynx were normal. The uvula was not

elongated. The nose was negative. The patient was passing through the menopause. She had an air of extreme nervous tension. Since the throat was negative, it was thought that the symptoms were of a reflex origin, and in pursuing this line of investigation I questioned the patient regarding her eyes. She replied that her sight was getting poor, and reading and sewing tired her eyes greatly. She also suffered from severe headaches and dizziness.

Examination of the eyes disclosed 1.50 diopters of hypermetropia and 1.25 diopters of presbyopia, for both of which she was wearing +1.25 sphere for reading only. Proper glasses were prescribed in bifocal form, to be worn constantly. The patient accepted this line of treatment for her throat trouble in a skeptical frame of mind. One week later, she reported that there had been only one mild attack of dyspnea, and that the other throat symptoms, together with the headache and many nervous symptoms which she had attributed to the menopause, had disappeared. At the end of six months there had been no recurrence of the throat symptoms.

Two years later the patient requested another examination of her eyes because the throat symptoms were returning. It was found that they had changed enough to require different glasses. This was done, and a few days later the throat symptoms disappeared. This experience was repeated again two years later. This would seem to prove that eye-strain may cause this condition, and to my mind it is its commonest cause.

Miss H., a school teacher, age 37 years, consulted me in April, 1919, for discomfort in her throat. Her description was vague but the chief sensation was that of a lump in her throat. She tired easily and was nervous. There were occasional headaches.

Examination of the throat showed small, flat, diseased tonsils. The anterior pillars were dusky red, the lingual tonsil was not enlarged. The nose, nasopharynx and larynx were essentially normal. The patient was somewhat underweight and pale and nervous. Examination of the eyes disclosed hypermetropia of 2 diopters, for which she was wearing only 1 diopter. She was told that the throat symptoms probably came from eye-strain, but since the removal of tonsils was

indicated by her anemia and lack of tone, she consented to the operation, supposing that it would effect a cure.

The tonsillectomy was performed on May 1, 1919. On June 2 the patient felt much better in general health but the sensation of a lump in the throat was still present. Her glasses were then changed. Two weeks later she reported complete relief from the throat symptoms and headaches and from the nervous tension which had been present for a long time. Twice during the last four years she has reported recurrences of the throat symptoms, each time obtaining complete relief from a change of glasses.

I have relieved many similar cases in the same manner during the last 15 years; more than enough, it would seem, to prove that eye-strain is a frequent cause of tinnitus aurium and globus hystericus.

PROGRESSIVE DEAFNESS—AN ABSTRACT SUMMARY OF THE AVAILABLE LITERATURE PUBLISHED DURING 1941.

DR. EDWARD H. CAMPBELL and DR. LOUIS E. SILCOX, Philadelphia.

The articles published in 1941 dealing with progressive deafness and its related subjects have rather closely followed the trend of those of the preceding year, dealing largely with the various aspects of otosclerosis, particularly its surgical treatment and experimental efforts concerned with this treatment. This review may appear to be incomplete because of the lack of mention of several articles that would seem to have some relation to progressive deafness. Some articles have been intentionally omitted because they have been largely reviews and have contributed no new facts or ideas regarding the subject. Other contributions published in certain of the foreign journals have not been available for review. Still other articles, while dealing to some extent with features of progressive deafness, have emphasized more particularly features pertaining to neuro-otology and the physiology and pathology of the middle and internal ear structures. Such contributions as these have been omitted. On the other hand, several papers published in foreign journals in 1940 have been included, since they were not available for inclusion in the abstract summary published the year before. In presenting these abstracts with the views and opinions of the authors, I have preferred to withhold any personal comment on their merits or demerits.

The abstracted material is arranged in the three groups employed by Dr. James A. Babbitt in preceding bibliographic summaries as follows:

Group 1: Clinical Studies and Research.

Group 2: Medical Treatment and Systemic Readjustment.

Group 3: Surgical Treatment.

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GROUP 1: CLINICAL STUDIES AND RESEARCH.

Important statistics concerning hearing defects in children were offered by Kerridge.[1] The author discovered that with the development of the gramophone audiometer, it has been possible to find that 80 children out of a thousand over 7 years of age in England have defective hearing.

The majority of defects of hearing in children are due to chronic middle ear disease — either suppurative or catarrhal.

Most of the chronic cases are found in the poorer classes. The resistance of the individual is the important thing early in these cases.

Complete deafness is very rare, occurring in about 3 per cent of children in schools for the deaf. Sound amplifying systems have been incorporated in most schools for the deaf.

The author stresses the fact that defects of hearing have not received the attention from public health workers that has been accorded to other physical defects.

Further statistics on the deafness of school children were produced by Emmerig.[2] The author undertook extensive research on the pupils of the public schools for the deaf in Munich. The research was limited to 237 cases, and these were sporadic rather than endemic. Causes of the deafness were tabulated as: Hereditary, 30 per cent; congenital, 27 per cent; acquired, 35.4 per cent; and doubtful, 7.6 per cent. Among the 237 cases, one-third, *i.e.*, 84, are acquired. The most frequent cause of this type is cerebral disease, mainly epidemic spinal meningitis. Another cause is auditory disease, most frequently labyrinth destruction. Other causes are primary labyrinth injury having middle ear complications, trauma, diphtheria, scarlet fever, mumps, etc.

In the author's opinion Emmerig's research is too limited to represent a statistical evaluation for establishment of etiologic viewpoints. The number of children afflicted with hereditary deafness is so great that, from the racial hygienic standpoint, the elimination of hereditably ill masses by means of sterility laws is justified.

Opinions on senile deafness were expressed by Saxen.[3] He stated that the clinical disease picture of senile deafness has

been thoroughly studied, but knowledge of the pathologic anatomy and pathogenesis of this affection is still incomplete. Typical symptoms of senile deafness are lowering of upper tone boundaries and diminution of auditory duration for high tones, and these symptoms appear comparatively early in life and become progressively more pronounced. There is always, too, decrease in the bone conduction.

In view of the clinical and pathologic-anatomic findings, many authors believe that the cause of this type of deafness lies in the labyrinth or in the acoustic nerves. On this, however, there is a difference of opinion. Sporleder asserts that in senile deafness the peripheral auditory apparatus is not affected, but that the cause must be sought in disturbances of the central auditory system due to injuries. He bases this view on the fact that in his histologic studies he found no instance where pathologic changes in the labyrinth or in the auditory nerves could be definitely established. O. Mayer propounds a causal theory quite divergent from all others thus far presented. He divides typical senile changes into two groups: those in which degenerative changes occur as result of characteristic senile diseases; and those in which presbycusis results from rigidity of basilar membrane. Mayer justifies his theory on the basis of having established thickening, homogenization and calcium crustation, as well as ossification of the membrane in the basilar membrane, especially in the proximal parts of the cochlear duct.

Regarding the changes that may occur in the labyrinth in otosclerosis, Saxen[4] gave the following opinions: Labyrinth changes, degenerative in nature, are often associated with otosclerosis. Little is known, however, concerning the interpretation of the pathogenetic association of the actual disease and the different parts of the ear. Whether it is coincidental, whether it can be explained on the basis of similar etiologic factors, or whether the otosclerotic bone foci cause secondary changes in the labyrinth are questions still unanswered. The last instance would be applicable only in cases where the labyrinth walls had been affected by otosclerotic processes. Atrophy of the nerves takes place, and it is quite conceivable that retrograde changes may occur in the spiral ganglion. O. Mayer observed that if bone formation extends deep into the cochlea, *i.e.*, into the wall between the basal and middle

convolutions, the blood vessels of the labyrinth may be obliterated, causing vascular engorgement in the labyrinth. As a result of this, he noted various types of labyrinth changes, such as transudation, edema, pigmentation, hemorrhage, as well as atrophy of labyrinth membrane. Even if direct development of foci in the intervening walls cannot be determined, the blood and lymph spaces of the cochlea may ossify and result in serious regressive changes in the walls of the membranous labyrinth, especially in the epithelia. Aside from these cases in which otosclerotic bone formation is considered responsible for labyrinth disease, there are many instances in which labyrinth changes occur independently and far removed from bone foci. The pathogenesis of this is still obscure. It has been suggested that they are a type of malformation, or that labyrinth atrophy is to be considered a form of hereditary degeneration. The author has nothing new to add to these theories. He had anticipated finding more frequent otosclerotic bone changes in his considerable amount of research material, but it included only one case of otosclerosis which was both clinically and pathologic-anatomically typical.

A timely article on auditory impairment due to the effects of war was presented by Schmücker.[5] He stated that auditory injuries due to war effects were formerly relatively rare. Today, however, because of enormous increase in the use of bombs, mines, grenades, etc., they have become extraordinarily frequent. In the World War, auditory injuries comprised 12 per cent of total casualties, and causes are classified as follows:

1. Impaired hearing due to effects of noise.
2. Impaired hearing due to detonation.
3. Impaired hearing due to shooting and intense noise.
4. Impaired hearing due to cranial injury.
5. Psychogenic disturbances.

Clinically, there is little difference between detonation injuries and sound injuries, since every detonation is accompanied by intense sound effects. Pathologic-anatomic results, too, are similar; the one exception is that tympanic membrane rupture is frequently noted in detonations but does not occur in pure sound injuries.

Tympanic membrane ruptures were of frequent occurrence in the first World War, and indications point to similar conditions in the present war. Symptoms in the vestibular parts of the auditory nerves are rare, and consist subjectively of vertigo, and objectively of nystagmus.

Prognosis of impaired hearing caused by sound and detonation is that in many cases improvement is noted after a very short time. Otosclerosis and progressive labyrinth deafness are found in cases where auditory disease had previously existed.

Prognosis of tympanic membrane rupture is favorable especially if proper and prompt treatment can be arranged; without this, however, the author observed numerous cicatrized ruptures which did not heal during the course of the war, and among these were perforations which included almost the entire tympanic membrane. In this connection it has been demonstrated that cleansing of the ear is a technical error, since it invariably results in middle ear suppuration, followed by mastoiditis.

Auditory impairment following effects of shooting and direct trauma is dependent on extent and site of the injury. Injury to the labyrinth and serious injuries in the middle ear cause permanent deafness. Injuries to the tube and auditory meatus may lead to more or less seriously impaired hearing. Head injuries resulting in pyramidal fractures may cause partial or complete deafness, depending on whether it is only a partial break, or whether the damaged cranial fissures extend into the bony labyrinth. The extent of deafness is largely dependent on the type of temporal bone fracture. Transverse fractures have much more serious effects on hearing than longitudinal fractures.

Psychogenic auditory disturbances may be so closely associated with organic hearing impairment that they are difficult to establish with any degree of certainty. Prognosis depends on early diagnosis and proper treatment. If functional auditory disturbance is suspected, the patient should not be told of the serious nature of the illness, at least not until his hearing has been restored. Efficacious methods of treatment are hypnosis, prolonged sleep (40 to 60 hours), prolonged bathing or treatment with strong faradic currents.

Good results, however, depend far more on the physician than on the methods employed. The will to cure is of paramount importance, and the inexperienced, interested physician frequently gets better results than the one who has the finest therapeutic equipment.

Asherson[6] theorizes on the causation of nerve deafness as follows: Nerve deafness, commonly occurring among the aged and called presbycusis, manifests itself by a loss of perception for high tones; and low tones in many cases being perceived normally. Thus, only a part of the cochlear nerve is involved. This suggests that the cochlear nerve is a composite nerve — one part for high and the other for low tones — the part concerned with high tone transmission being the more vulnerable of the two trunks. The author presents a theory that the lesion involving the cochlear nerve is never primary but is secondary to vascular occlusion. The blood supply may be shut off by spasm, embolus, thrombophlebitis obliterans, endarteritis — such as is seen in syphilis or associated with arteriosclerosis. When an end-artery is blocked, complete permanent nerve damage is done.

He suggests that otosclerosis may be on a vascular basis, the process first obliterating the blood supply to the low tones and later to the higher ones. He cites a case of nerve paralysis on a vascular basis.

A review of various facts and theories concerning senile deafness was contributed by Hofer.[7] For many years senile deafness has been the subject of exhaustive study by otologists, with a view to determining its particular type of deafness. Early research consisted of functional tests, and in later research anatomical tests were made on the labyrinths. Sporleder tested the auditory organs of 100 patients ranging in age from 50 to 90 years. Results showed that, beginning at 50 years of age, the auditory facilities steadily diminished, and the grades of impaired hearing were equally distributed in both sexes. Sporleder was the first to make postmortem studies of the temporal bone of deaf patients. In no instance could he definitely establish serious disease changes in the labyrinth — either atrophy of the cochlear nerve or changes in the labyrinth capsule; therefore, he concluded that the cause of senile deafness could not lie in the labyrinth itself, but rather should be sought in the cen-

tral auditory nerve system. This research was of great moment since it brought the study of senile deafness out of the purely clinical stage into the stage of anatomical research.

Concerning the prognosis of senile deafness, the efficacy of hormone therapy is yet to be determined. Thus far, results consist in disappearance of subjective auditory symptoms, and in improvement of hearing in a limited number of cases. This improvement persisted in some cases for one to two years, and sometimes even for a longer period. Further research with hormone therapy may have far-reaching results, especially if the changes already existing are not too serious.

Holmgren[8] considered that the symptoms in the external and middle ears in otosclerosis have not been sufficiently considered and no attempt has been made to bring them into alignment with the fundamental condition. Most of the textbooks hardly mention these symptoms. Reduction of the secretion of cerumen and of the sensibility of the skin are well known, but the other symptoms are rarely mentioned. They include atrophy of the skin of the meatus, dilatation of the lumen, reduction of the tickling reflex, thinness and transparency of the drum, and reduced or absent motility of the handle of the malleus, and increased mobility of the tense portion of the membrane, and the loss of vascular reflexes, and the author considers them all to be symptoms of ankylosis of the stapes.

The author's theory is as follows:

An increase of the intralabyrinthine pressure fixes the footplate of the stapes and predisposes to ankylosis, then all the ossicles of the chain become fixed. When the handle of the malleus is immobile, the vascularity of the tympanic membrane and wall of the external meatus is changed either mechanically or reflexly with atrophy and a reduced irritability as consequences.

Ersner and Saltzman[9] presented the case reports of three cases of familial deafness involving 50 per cent of the females of three successive generations and affecting first the right ear in each instance, while all the male members of the family possessed normal hearing. They considered that these cases constituted an illustration of progressive analogous nerve deafness with sex-limited inheritance. They considered

that these patients had an affection which is related to otosclerosis but is not otosclerosis.

An article, devoted largely to an analysis and criticism of the labyrinth fenestration operation in general, and Lempert's technique and results in particular, was contributed by Katz.[10] He calls attention to a number of obvious discrepancies in the report of Lempert's early cases. Katz advances a theory for the improvement of hearing in fenestration operations, which he calls the theory of "suction action." He explains that the escaping perilymph in the labyrinth (through the fenestrum) creates a vacuum, with the result that more blood is brought to the labyrinth, causing an improvement of hearing.

Crowe and Burman[11] have given reports of the continuation of their study and treatment of deafness in children by radiation. During the past 16 years they have made hearing tests on more than 15,000 patients, about 3,000 of whom were children. They have drawn the following conclusions:

1. Impaired hearing for high tones with good hearing for low tones is the earliest symptom of middle ear, as well as inner ear deafness in children.

2. The recurrence of adenoids after operation on young children is so common that it must be regarded as normal. This lymphoid tissue may impair the function of the Eustachian tube, leading to chronic progressive deafness.

3. Every type of deafness seen in adults is also found in children. In most types the earliest symptom is a loss for high tones, particularly above 8,000 d.v.

4. Early recognition of the impaired hearing and radiation of the nasopharynx to restore the patency of the Eustachian tubes is the only method for the prevention of progressive deafness of this type.

5. The dosage of radiation in such cases is far below the amount that could cause any injury to the adjacent tissues. The type of radiation recommended is radon placed in a nasal applicator to come in direct contact with the area of the Eustachian orifice.

6. Radiation is of no value in the treatment of chronic middle ear suppuration, otosclerosis or any form of inner ear or nerve deafness.

In an article reviewing many of the well known features of Ménière's syndrome, Grove[12] mentioned several of the theories concerned with its etiology, reviewed the symptomatology and diagnosis of the condition and stressed some features of the treatment. He felt that in the etiology more serious consideration must be given to allergy, avitaminosis and disturbances in the endocrine balances, and thought that the main pathological feature of the condition is a waterlogging or edema, as maintained by Mygind, Dederding, Furstenberg and others. He suggested that this waterlogging process may be operating both in the end-organ and in the central cochlear nuclei, which might explain the persistence of tinnitus after the nerve has been severed. He believes that before submitting to surgical intervention, the dehydration treatment of Mygind and Dederding, the sodium elimination treatment of Furstenberg, and the treatment with histamine should be thoroughly tried.

Hughson and Thompson[13] have presented what they call a critical summary of a 10-year period in otological research. Their historical account indicates that the physiology of hearing has been established upon a rational and fundamental basis, particularly on the basis of the discovery of the relationship of atrophy of the end-organs in a particular part of the cochlea, with loss of certain frequencies and the discovery of the electrical changes in the auditory apparatus initiated by sound impulses. They believed that endocrine, vitamin and drug therapy of deafness had been exploited far beyond their proven value, but that they offered problems for research both in the matter of prevention and alleviation of deafness.

The surgery of deafness also received consideration, as well as experimental work concerning the production of labyrinthine fistulae and the preservation of such openings. They reviewed in some detail the newer diagnostic methods including the use of audiometers, masking, loudness balance test and the fatigue test, which have led to the exact diagnosis of deafness and accurate measurement of hearing loss. The rapid development of hearing aids was discussed and it was thought that they would soon be on a more standardized and practical basis.

Newhart[14] reviewed the progress that had been made in the past few years in the conservation of hearing. He empha-

sized the value that the audiometer has been in the detection of early slight and frequently unsuspected deafness. He believed that a practical plan for conserving the hearing would involve legislation providing for readjustments of the activities of various educational public health, welfare and social agencies and for increased appropriations. An important step would be the incorporation in every school health program of periodic testing of the hearing of all pupils by the audiometer. He believed greater initiative and leadership by otologists and interested civic-minded citizens was needed to carry on an organized educational program for conserving the hearing, which would result in effective legislation.

A profound treatise on tinnitus aurium has been contributed by Fowler.[15] He has investigated the subject exhaustively and summarized his findings in part as follows:

1. Tinnitus can be divided into two fundamental types, vibratory and nonvibratory. The first is mechanical in origin, due to actual vibrations. The second is biochemical in origin and not due to any actual vibrations.

2. The loudness of tinnitus may be objectively estimated by comparing it to an applied tone or noise of known loudness.

3. There may an "illusion of great intensity" of tinnitus in which the patient thinks his head noises are very loud, but actually are faint. The loudness balance technique can determine this.

4. The methods of estimating the tone or frequency band of tinnitus and the masking of tinnitus by discrete and by band frequencies were discussed.

5. Neither the loudness of the tinnitus nor its frequency alone determined the ease of masking it.

6. The presence of tinnitus is always associated with more or less deafness.

7. The frequency and loudness of tinnitus determines the deafness caused by the tinnitus lesions.

Several other features of tinnitus, with discussions on their significance, were brought out by Fowler. The article contains much interesting and instructive information which undoubtedly will be valuable as an authoritative reference.

Further experimentation along the lines of the possible relationship between Eustachian tube obstruction and deafness of children was conducted by Mao and Hughson.[16] They examined 331 children with considerable deafness to determine whether or not the Eustachian tubes were patent within relatively normal limits. They used a method of autoinflation and considered 60 cm. of water a pressure requirement beyond which function of the Eustachian tube could no longer be considered normal. The children ranged in age from 9 to 21 years. Adenoid tissue near the Eustachian orifice was seldom found and when present was apparently not an important factor in relation to tubal patency. They found no apparent relationship between biologic deafness with profound loss of hearing and inflatability of the middle ear. Even thickening and retraction of the drum membrane were not particularly frequent in the patients whose tubes failed to inflate.

The psychology of deafness in children was studied by Drennan.[17] He states that medical literature with reference to deafness and the psychological reactions associated lends very little assistance to a study of these reactions in a school for the deaf where 70 per cent of the 500 to 600 pupils are under 15 years of age.

He uses two groups to classify deafness in children:

The Deaf or those in whom the sense of hearing is nonfunctional for the ordinary purposes of life, and these may be either congenitally deaf or those who lose functional hearing later.

The Hard-of-Hearing or those in whom the sense of hearing, although defective, is functional with or without a hearing aid.

Often a child does not know that he is deaf, and very often thinks he is stupid because he does not get along as well as other children. It is an unending struggle to keep up with conversation, and in many the struggle goes beyond the capacity of the patient to sustain himself, and he gives up and becomes unhappy and apathetic. The deaf child feels isolated, and he becomes depressed and feels persecuted.

The development of speech in the congenitally deaf is done by a very tedious artificial process. The speech is entirely

different from that of normal children who learn to speak by hearing others.

Ruth Brickner states that there is a psychiatric principle that under unbearable emotional stress, an individual will break down in the direction of the weaker supports of the psychic structure. Everyone dealing with the deaf is familiar with the emotional explosions, and there are many who by repeated emotional explosions end up with a complete nervous breakdown or insanity. The author stresses the fact that many cases of deafness in children now can be corrected and many more can be prevented by proper application of the newer preventive measures for deafness in children.

The effect of noise on aviators was discussed by Pastore.[18] He found that during the flight the hearing of the aviator may be affected by a number of noise factors. Sounds caused by the motor, propellers, exhaust, wind, radio signals and loud talking are some of the common noises. During war, gun and cannon fire are additional sources of noise. Several articles have appeared in the literature on this subject and one points out that deafness in aviators may be classified according to four causes:

1. Acute fatigue.

2. Chronic accumulative fatigue.

3. Changes in pressure in the middle ear.

4. Chronic conductive deafness caused by tissue alteration which results from faulty ventilation of the middle ear.

This report concerns audiographic records of 88 of 100 experienced pilots employed by one of the major commercial airlines of the country.

Age range, 22 to 40 years.

Period of flying time, eight months to 17 years each.

Only one acute otitis media had been present in this group.

Five of the pilots had so-called borderline normal hearing.

Forty-two of the pilots had normal hearing.

Sixteen of the pilots had nerve deafness.

Three of the pilots had unilateral conduction loss.

Twenty-two of the pilots had conduction type loss.

Results indicate that pilots are not equally affected by exposure to noises caused by airplanes. Two pilots each had a record of 14 years of active flying service, yet each had normal hearing by bone and air conduction audiometer test. Among the 22 pilots with air conduction type of deafness, the zone of greatest loss was in zone of 4,096 cycles; note: ear protection for aviators has been suggested.

Further researches on acoustic trauma caused by warfare were contributed by Rüede.[19] His opinions and suggestions are as follows:

The two forms of acoustic trauma may be differentiated as, in *1.* so-called "chronic" acoustic trauma, which corresponds intrinsically to sound injury; *2.* "acute" acoustic trauma, which some authors believe should be more accurately termed "explosion" trauma.

The prototype of chronic acoustic trauma is the occupational deafness of the coppersmith. Following long-continued noise peculiar to his craft, the majority of workers sooner or later are afflicted with labyrinth deafness. In animal experiments, Wittmaack showed corresponding injuries by the use of whistles and sirens. These were followed by circumscribed histologic changes in the cells of Corti's organ, location of injuries, depending on pitch and frequency of tones.

Three tables show minutely detailed findings in acoustic injuries caused by small calibre weapons. These indicate wide variations of labyrinth deafness frequently following intact tympanic membrane. The cause of these individual variations, however, should be attributed not so much to the mechanical destructive explosions but rather to effects on the injury process of hitherto little noted sound waves of high intensity. Peculiarities observed in the clinical picture can undoubtedly be explained by this hypothesis.

The author suggests four prophylactic measures:

1. Detailed auditory research on all recruits in order to eliminate certain types of deafness at the beginning.

2. Otologic control of all acoustic trauma occurring during military service, even the quickly healing types.

3. Employment of only those with perfect hearing in the attacking divisions; *e.g.*, in the infantry, where cannon are used.

4. Conduction of sound resistance tests, including tests for acoustic liability to fatigue. Similar tests are used in Russia and Sweden in cases of young people who elect to learn a trade, which guarantee against acoustic injury.

Tobeck[20] presented valuable statistics on ear injuries caused by war. It is significant that in time of war, illnesses and injuries occur which are rarely, if ever, noted in peace times. In the field of otology, detonation is the principal cause of injuries peculiar to war periods. In the first World War, traumatic labyrinth injury with tympanic membrane rupture was the most frequently encountered auditory injury. There is every reason to assume that the present war will show similar findings.

A study was made of 200 cases of auditory injuries in World War I, and results obtained in 187 cases are indicated in the following tables:

TABLE I.

Causes of Tympanic Membrane Injury	Number	Percentage
Exploding grenades	92	49.21
Obstruction due to exploded grenades	11	5.88
Exploding mines	37	19.80
Obstruction due to exploded mines	15	8.02
Hand grenade explosion	16	8.55
Cannon fire	10	5.35
Explosions	4	2.13
Exploding shrapnel	1	0.53
Exploding aerial bombs	1	0.53

TABLE II.

Group	Type of Injury	Percentage
I.	Unilateral injury without perforation	14
II.	Unilateral injury with single perforation	33
III.	Unilateral injury with several perforations	3
IV.	Bilateral injury without perforation	9
V.	Bilateral injury with perforation	41
VI.	Bilateral injury with single perforation	62
VII.	Bilateral injury with single perforation on one side and several perforations on other side	21
VIII.	Bilateral injury with perforations on both sides	4

Other tables indicate that in the occurrence of tympanic membrane perforation the anterior portion is more frequently

involved than the posterior. All grades and types of auditory disturbances due to detonation were studied, and the most frequent was marked reduction of auditory capacity for whispering, with consequent mixed hearing impairment. Injuries to the auditory meatus involve mechanical rather than acoustic trauma.

Since observations on these 187 soldiers were made only once, and that was during the first 10 days after the injury, it is not possible to comment on the healing of the tympanic membrane perforations nor on the later conditions of the auditory apparatus.

The pathologic involvement in otosclerosis was commented upon by Nager[21] as follows:

In one of his works, Holmgren[8] demonstrated that in several cases of otosclerosis the handle of malleus remained fixed on application of Siegle's pump, while other parts of the tympanic membrane followed along normal lines after each pressure change in the auditory meatus. Study of a series of auditory ossicles in otosclerosis revealed more or less significant changes of articulation between incus and stirrup bone and of malleus. These are attributed to shifting of stapes laminae and react by means of stretching of the joints, being dependent on expansion of the processes in the oval window niches. In very pronounced cases, far-reaching changes occur, even tightening in the malleus incus joints. These changes may be due to the inflexibility of malleus. In addition to this, the substance of the auditory ossicles showed bone changes similar to those observed in otosclerosis. In one case there was found marked otosclerosis of the greater portion of malleus and incus. The appearance of the disease at these sites is explicable in that they, like the labyrinth capsule, are typically cartilaginous and are not completely surrounded but consist predominantly of plexus type and strand bones in the interglobular spaces of which cartilage residue can be found.

Dickson and Ewing[22] describe recent experiments to ascertain how far devices at present available to the fighting service and general public afford protection to the sense of hearing. Tests have been made during flight, in the laboratory and by engineers testing aircraft engines on the bench. Com-

plete elimination of the risk of deafness can be assured only through occlusion of the meatus, combined with efficient covering of the ears. The investgations show that noise components of middle and low frequency may be responsible for numerous cases of occupational deafness observed among flying and ground personnel.

The first symptom of occupation or traumatic deafness is usually a loss of acuity to sound of frequency about 4,096. This may be because response to such sound is greatest in the basal turn of the cochlea adjacent to the middle ear, which bears the brunt of stimulation by noise due to explosions.

In a contribution on Ménière's syndrome, Atkinson[23] expressed his views on the classification and etiology of this condition, as well as the vestibular function and its mechanism of production in such cases. The basis of his theory is that Ménière's syndrome attacks are brought about by sudden changes in the blood vessels in the vestibule and can be produced by either of two mechanisms, the one allergic and the other vasospastic, which can be differentiated by the histamine skin test. In the allergic group, vestibular function was either diminished or unchanged, while in the vasospastic group it was almost invariably increased. It was also found that in the former group there was usually a bilateral conductive deafness, while in the latter group perceptive deafness was found. Atkinson[23] states that in the allergic group there is an increased output of endolymph which dampens down the movements of the hair cells in the semicircular canals on the affected side until function is so diminished that vertigo results. The same dampening-down process affects the cochlea, with diminution of hearing to all tones. The modus operandi of the vasoconstrictor group is supposed to be just the reverse, the constriction of the vessels causing less pressure of endolymph, resulting in an imbalance which produces vertigo. The decreased blood supply to the cochlea would explain the perceptive deafness present in these cases, according to Atkinson.[23]

As a result of their experience, Ersner and Myers[24] were of the opinion that the heat of a burnishing burr in the production of a labyrinthine fenestrum does not inhibit osteogenesis. They measured the temperatures on the surface of the external semicircular canal of a cadaver, on the surface

of the occipital bone of a cat, and the intralabyrinthine temperature of a cat, both before and after the use of the burr. The smooth, dull burr did not give an increase in the heat beyond 3° F.

Fowler, Jr.,[25] contributes considerable information on the mechanisms of bone growth in the labyrinthine capsule as they occur in monkeys when an artificial fistula is made in the horizontal semicircular canal. His beautiful histologic illustrations give definite evidence of the truth of the conclusions he has drawn regarding his experimentation. He briefly reviews the experimental work, both on animals and humans, on the postoperative reaction of the fistula that has been done by Canfield, Meltzer and the Guggenheims, Holmgren, Nager, Sourdille and Lempert. He proves by measurements the impossibility of covering the fistula in the external semicircular canal with Shrapnell's membrane. From his experimental work in the production of external semicircular canal fistulas in seven large monkeys, Fowler[25] has drawn the following conclusions:

1. Closure of labyrinthine fistulas in monkeys is almost the rule, whether they are made with a polishing burr or with a curette, and yet bony closure of fistulas made with either instrument may not occur.

2. The periosteal bone of the monkey seems to grow even more rapidly and more constantly than that of human beings.

3. The most marked growth is from the periosteal capsule and its periosteum, but growth also occurs from the endosteum and probably from the edge of the endosteal capsule. Growth from the endochondrial layer has also been observed.

4. There is always some osteoid tissue about any bone chips.

5. The presence of epithelium reduces regeneration of bone.

6. The use of Thiersch grafts is fraught with the danger of producing epidermal inclusion cysts.

7. The reason why hearing is better in human beings when the incus is left in place is that the incus holds the skin flap sufficiently away from the fistula so that cuboidal epithelium on the back of the flap can grow backward and medially to meet the epithelium from the front of the horizontal semicircular canal as it grows backward and laterally.

8. The monkey's mastoid cavities in which infection developed showed much faster regeneration of bone than the cavities which were relatively sterile.

9. The localized labyrinthitis present explains the vertigo of patients who have been operated upon, and also why their reaction to the fistula test for vertigo tends to become less positive a few days after operation.

Further animal experimentation by the Guggenheims[26] regarding the inhibition of osteogenesis on artificial bone fistulas reveals interesting results. They made fistulas in the craniums of white rabbits by means of a sharp chisel and treated the osseous fistulas with copper. The experiments were made on the contention (as stated by the Guggenheims) that copper, even in minute amounts, inactivates vitamin C by catalytic oxidation. As vitamin C is necessary to the jelling of collagen in the formation of bone matrix, bone cannot form in the absence of vitamin C. In the one experiment which was completed, the osseous fistula was treated with saturated solution of copper sulfate and a fine filament of copper wire was tied around a projecting portion of bone. After two months and 11 days the specimen was removed and the fistula was found free of osteogenesis.

Experimental work having a possible bearing on the treatment of otosclerosis was presented by Guggenheim, Gunther, Goodhill and Irvine.[27] Their observations had shown that classical otosclerotic patients treated with dietary regulation and administration of dicalcium phosphate, vitamins, insulin, extract of liver and other preparations and observed over a period of four years had shown no loss of hearing, and some even showed an improvement of hearing. They believed a possible explanation of the absence of further deterioration in hearing might be found in the reversal of halisteresis in the aural capsule — a rebuilding of osseous tissue around the focus of otosclerosis. Such a reossification would tend to inhibit further increase in the size of the dystrophic area of otosclerosis. Their experiments were undertaken in an attempt to prove the possibility of reversing halisteresis. Decalcification of the aural capsule was produced in Wistar white rats with parathyroid extract, viosterol and ammonium chloride. With a second group of animals, ammonium chloride was used to produce decalcification, after which a rever-

sal of the process (osseous reconstruction) was accomplished by removing ammonium chloride from the diet and placing the rats on a high calcium diet. They concluded that it seems possible clinically to inhibit enlargement of an otosclerotic focus with adequate amount of calcium, phosphorus and vitamins which appeared to cause a reconstruction of capsular bone in the decalcifying areas around the dystrophic focus.

Hagens[28] reports the occurrence of otosclerosis in identical twins, in which the deafness of one twin increased over that of the other. The only known factor to account for this was that the twin with the poorer hearing had a child.

Experimental work in auditory fatigue was conducted by Kobrak, Lindsay and Perlman.[29] This was based on the acoustic reflex of the muscle of the middle ear, which offered a quantitative method of studying the acoustic stimulation in the cochlea independent from the cortex. Observations were made on animals and on the human. The acoustic reflexes and hearing sensations were observed under the influence of a fatiguing tone. The following is their summary of these experiments: "There are observations which show subjective fatigue without signs of diminished cochlear irritation. This fatigue is interpreted as central fatigue. The second group consists of experiments which show an elevation of the auditory threshold, both objectively and subjectively. This kind of fatigue is located either in the cochlea or in the primary acoustic nuclei. It is morely likely that the seat of this fatigue is the cochlea. The third group of experiments consists in observations of the reflex contractions during the prolonged stimulation. Observed is a slow relaxation of the muscle contraction. This relaxation is different from true fatigue, since recovery symptoms observed in true fatigue are missing. This phenomenon may be interpreted as acoustic adaptation. The observations made in these experiments are therefore grouped as central fatigue, peripheral fatigue and auditory adaptation. The acoustic stimuli of daily life do not fatigue the acoustic reflex of the muscles of the middle ear."

The effects on the ear of deficiency of vitamins has been discussed by Covell.[30] He states that it has been proven experimentally that diets lacking or low in vitamin A cause an increase in thickness of the periosteal layer of the otic capsule sufficient to form circumscribed areas (exostoses) in

the internal auditory meatus and modiolus; diets lacking or low in vitamin C cause a replacement of the cellular marrow by that of the fibrous type in the periosteal layer; avitaminosis D produce characteristic bony lesions in the periosteal and enchondral layers; and avitaminosis E is associated with irregular thickening of the periosteal layer. The different parts of the internal ear, including the specialized sensory or hair cells of the organ of Corti have been shown to be altered in vitamin deficient animals. Changes in the VIIIth nerve and its ganglion cells (both cochlear and vestibular) result from deficiencies of the vitamin B complex and may occur in lesser degrees in other deficiencies. The author states that periods of months, and even years, are necessary for the actual repair of nerve fibres; and for this reason no immediate recovery of the VIIIth nerve can be accomplished by vitamin therapy.

Smith[31] brings to our attention the subject of von Recklinghausen's Disease in Otology. He reports a case with typical pigmentary change, subcutaneous fibrous tumors along the course of a nerve, a large collection of typical neuronomata in the temporal region, and a meningocele. He states there are numerous reports of neuronomata attached to both auditory nerves in von Recklinghausen's disease. These auditory tumors comprise 6 per cent of all cranial tumors. Otorrhea may be an etiological factor. They are commonest in the fifth decade of life. The first symptom is tinnitus. Impaired hearing accompanies it and gradually becomes worse. The vestibular branch is more often completely ablated than the auditory branch. Unsteady gait and dizziness soon occur and discomfort in the occipital region is next noticed. As the growth enlarges, extreme tenderness of the occipital muscles occurs and cerebellar symptoms become more manifest. The adjacent cerebral nerves may next be affected and give characteristic symptoms.

Through a survey of the literature, Jones[32] has discovered some unusual pathological findings in otosclerosis. He states that the most common site of an otosclerosis is in the region of the oval window. Less common locations are the first turn of the cochlea causing exostoses into the labyrinth or extending into the middle ear cavity, the bony spiral lamina of the cochlea, Corti's organ, the area of the round window and the modiolus, the semicircular canals, the vestibule and the Fallopian canal causing a facial paralysis.

GROUP 2. MEDICAL TREATMENT AND SYSTEMIC READJUSTMENT.

An opinion of the value of vitamins in deafness and tinnitus was expressed by Baer.[33] He agrees with Szolnoky that vitamin C is often effective in bringing about the disappearance of tinnitus aurium, and, like Szolnoky, he, too has had cases where it failed with no definite explanation. There is as yet no known method of curing tinnitus aurium; therefore he believes vitamin therapy holds out some hope and can be favorably regarded in spite of contraindications.

A short time later, Baer[34] expressed his ideas on vitamin therapy of otosclerosis as follows: "Thus far, vitamin therapy has resulted in improvement of psychic symptoms of headache and tinnitus aurium." The author's treatment of patients extended over a period of four to five weeks, and consisted of massage and auditory diathermy followed by vitamin therapy. Only very rarely was there any improvement in auditory facilities. He then decided to concentrate on the possibility of vitamin E therapy in otosclerosis, but the prognosis has been exceedingly poor. Nevertheless, he maintains that since vitamin E medication is harmless, he will be justified in conducting further research with this vitamin, possibly in combination with hormone preparations. The results of his studies will be given after a proper amount of research has been completed. A further report on experimental work in the vitamin therapy of otosclerosis was contributed by Baer.[35] With regard to vitamin therapy in otosclerosis, extensive study with vitamins C and B_1 brought no results except disappearance of headache and tinnitus aurium symptoms in some cases. Literature on vitamin E allows of no E vitaminosis but invariably can attribute effects to various causes, principally neuromuscular injuries and changes in the labyrinth capsule. Since, in addition, there are no contraindications, the author decided to conduct experiments with this new vitamin in order to determine its efficacy. For his project he selected 10 otosclerotic patients, some of whom he had previously treated with vitamins C and B_1. Five of these cases were individuals over 40 years of age, with otosclerosis of long-standing and progressive in nature. Among the others was a youth of 19 years, who showed very slight improvement, and a boy of 14 years, who showed no improvement in hearing, probably due to concurrent operation for adenoids. The last three cases showed marked improvement.

They were, respectively, a youth of 17 years, who noted improved hearing in both ears, along with disappearance of tinnitus aurium; a child's nurse of 24 years, who reported improved hearing facilities and disappearance of tinnitus aurium after four weeks' treatment.

The author believes that while 10 cases may seem a small number, nevertheless results should be evaluated on the basis of the fact that otosclerosis is a comparatively rare disease. He thinks that the efficacy of vitamin E in young patients should not be underrated, since here it might at the very least retard the progress of otosclerotic processes. His failure to obtain results in two of the otosclerosis patients may probably be attributed to hormone disturbances. The daily E requirement is still unknown, as is also the E tolerance of the various organs of the body. Although this limited research leaves many questions unanswered, and has not produced outstanding results, the author is convinced that, due to poor prognosis of otosclerosis and in the futility of earlier therapy, further research with vitamin E is greatly to be recommended.

Ersner, Rush and Myers[36] reported the results of their treatment of 59 cases of deafness and tinnitus with prostigmin methylsulfate. Of these patients, conductive deafness was present in 10, nerve deafness in 10, otosclerosis in 14, and a mixed deafness in 25. The hearing measurements were done with calibrated audiometers. Each patient received a minimum of 35 injections of prostigmin-methylsulfate hypodermically (1 cc. of 1:2,000 sterile solution). Of the 59 patients, 44 per cent failed completely to show any change either subjectively or objectively, 6.7 per cent said that some improvement had occurred (not substantiated by audiograms), 30.5 per cent showed a subjective transitory improvement, and 13.5 per cent had audiometric evidence of an 8 to 10 dcb. improvement, which was also transitory. They concluded that prostigmin had little, if any, value in the treatment of these patients with deafness and tinnitus aurium.

Selfridge's[37] article on the relationship of vitamins in deafness deals largely with a summary of the symptoms and conditions found in a deficiency of the various vitamins and the correction of such conditions by dietary treatment. He repeats his contention that deafness is not caused by any

one thing but is related to all the factors involved in growth; that is, endocrine products, electrolytes, amino acids and vitamins. He states that the time to begin the prevention of deafness is six months before conception of the child. emphasizing the necessity of proper diet, particularly vitamins of the parents, and the dietary supervision of the infant and child.

At variance with a number of previous articles indicating no value in the treatment of deafness and tinnitus with prostigmin is the report of Rosenthal[38] on its use in 30 cases. Twenty-four had had loss of hearing over a long period of years, five had had impaired hearing for periods not longer than six months, two of which had only recent involvement. The type of impaired hearing appears not to have been mentioned. Twenty-three of the patients had associated tinnitus. The hearing was measured by a calibrated audiometer. An intramuscular injection of 1 cc. of a 1:2,000 solution of prostigmin methylsulfate was given three times a week. For children, a dose of 1:4,000 solution was used. In these cases the extension of the treatment varied from about one month to about six months. The results indicated that 19 patients, six with acute and 13 with chronic impairment showed objective improvement of hearing; 16 showed subjective improvement, and nine showed no improvement. Two patients reported that the condition had become worse. Sixteen said that tinnitus had disappeared or diminished, while three reported no abatement of tinnitus. The author concluded that prostigmin seems to have a definite value in impaired hearing of short duration and in some forms of tinnitus aurium.

Furstenberg, Richardson and Lathrop[39] reported on the results of their treatment of Ménière's disease during the past seven years. They carefully analyzed the results in 35 patients from the symptomatic standpoint. Their treatment had been based on the conclusions reached after considerable study, that the tissues responsible for Ménière's disease were sensitized or had an increased avidity for the sodium ion. The treatment, therefore, consisted in prescribing a sodium-free diet and the administration of ammonium chloride, which insured the maximum elimination of sodium in the excretion fluids. They had treated over 150 patients with Ménière's disease by this method with uniformly satisfactory results.

In their discussion of the etiology and pathology of the condition it was mentioned that the precise etiologic factors in Ménière's disease had not been determined, nor the location or the character of the lesion responsible for this entity been identified, although they were in accord with the views of Crowe and Hallpike and Cairns that the pathologic changes occurred in the vestibule and cochlea, and that either chemical or pressure changes of the endolymph, and possibly the perilymph, are responsible for the sudden attacks of vertigo.

In reviewing the treatment which is advocated for this condition, the authors emphasized the importance of the strict salt and sodium-free diet, the avoidance of drinking water containing sodium chloride, the administration of ammonium chloride in large doses (3 gm. with each meal for three days, then omitted for two days) for six weeks, and the necessity of hospitalization at the start of the treatment. They stated that the effect of the treatment is only relief from vertiginous attacks. The noises in the ears occasionally subsided but the deafness was usually progressive. Surgical intervention was resorted to only when an adequate trial of this medical therapy was fruitless.

In a further contribution, Atkinson[40] discussed in more detail his observations on the etiology and treatment of Ménière's syndrome, basing his opinions on his theory that the characteristic syndrome is the result of a vasomotor disturbance, which in one group of cases is allergic in origin and in the other is due to a vasospasm. In his paper he attempted to demonstrate the validity of this hypothesis, gave details of the histamine skin test, and mentioned the results of his clinical experiments and formulated clinical characteristics of the two groups.

In the treatment of those patients in the allergic group it was stated that the ideal treatment is to discover and remove, or desensitize against, the specific allergen. In seven patients the treatment was by histamine desensitization, starting with 0.01 mg. subcutaneously and working up in slowly increasing doses twice or at most three times weekly to the limit of tolerance, which has been as a maximum 1 mg. and sometimes less. They were then maintained at top dose once a week for a month. A second short course after a three months' interval is advisable. According to Atkinson, the

results have been most satisfactory. Headaches are relieved or disappear, the general condition improves considerably, but the deafness and tinnitus were not so uniformly improved.

In the vasomotor group, the author, after experimenting with various drugs, found that vitamin B gave the best results, especially the nicotinic acid fraction. He advocates the administration of nicotinic acid and thiamine hydrochloride to the limit of tolerance, maintain the dosage there as long as necessary, and then gradually diminish the dose to a maintenance dose which may be oral.

In a summary of the present status of otosclerosis. Babbitt and Silcox[41] discuss briefly facts concerning the etiology and pathology of this condition, emphasizing the rôle that vitamins, the diet and calcium and phosphorous metabolism may have in its production. They reviewed some of the work leading up to the present status of the surgical treatment of otosclerosis. Other types of treatment were also mentioned, such as various drug injections, vitamin, endocrine and dietary treatment. They were of the opinion that fenestration of the labyrinth has demonstrated possibilities of hearing recovery and that "the trial balance on the vitamins, endocrines and newer biochemical agents is not yet complete."

In a discussion on the psychological aspects of deafness, Frey[42] stated he had learned from the literature that among deaf-mutes the uneducated often appeared queer and not easy to handle, but that the educated ones were often careful, vigilant, could concentrate well and do good work in many trades. He thought that most deaf-mutes were not quite normal apart from their hearing defects, that the congenitally deaf were degenerate individuals showing abnormalities of their central nervous system, of their intelligence and of their psychical disposition, as well as some somatic stigmata. Where, however, deafness was acquired in early life, it could be assumed that the central nervous system had not survived the strain of the disease such as meningitis, without lasting inferiority of function. He was of the opinion that the deafened as a rule overestimate the extent of their deafness, and many overestimate the practical consequences of it. The otosclerotics represented a special type of a highly nervous character and highly unstable vasomotor conditions.

During the discussion of this subject, Mr. J. R. Ewing stated that the psychological effects of deafness, in addition

to deafness itself, depended upon such facts as health and age of the patient, his environment and experience, his social attitude, his disposition to difficulties, his intelligence, nature of his work, his resourcefulness and his sense of humor.

Hughson, Ciocco, Witting and Lawrence[43] presented interesting and informative data on speech characteristics of deafened children and on training methods. In their summary of this study the following information was elicited:

1. The speech characteristics of 366 children were studied. Of this group, 248 had never received auricular training at the time of the study, and 118 were enrolled in auricular classes.

2. In general, comparison of the two groups shows that the speech of children given auricular training tends more toward that of the normal child than does that of the oral group.

3. Regardless of age of onset, degree or duration of hearing loss, the auricular teaching method is superior to the oral for all children with any residual hearing.

A survey of deafness in school children by Reinking[44] brought out some interesting information and suggestions for treatment. In Germany, Russia and the United States, more than 20 per cent of school children have been found to have imperfect hearing. In Mexico the number is probably less, but still considerable. Parents are advised to secure medical treatment for children with impaired hearing, but in many parts of the country, particularly in rural districts, this is not possible. In many instances no treatment can effect sufficient improvement to enable the child to attend an ordinary school.

Reinking states that among 582 children in Lucerne schools, Nager found 154 with poor records. Of these, 23.2 per cent had slight deafness, and 34.3 per cent, more severe deafness. In Dortmund, Hansberg examined pupils who had failed and found that 53 per cent had defective hearing. In Rome, Mancioli found that most pupils with defective hearing had poor records, some got along well but none had excellent records.

In instruction of partially deaf children, Reinking considered the following points important:

1. Small classes (6-12).

2. Re-education of the ear, which is actually a development of intelligence and perception rather than of the ear itself.

3. Correction of speech defects, very frequent among children with impaired hearing.

4. Instruction in lip-reading.

Experimental work on the hormone therapy in deafness was reported by Perwitzschky and Naseman.[45] Although hormone therapy is not new, and in spite of the fact that all preparations of this type have yielded only negative results, the authors, at Dr. Zajicek's earnest request, consented to make thorough tests with his hormone ointment.

Under the personal instruction of Dr. Zajicek, careful experiments were conducted on 33 patients. These represented:

Six cases of labyrinth deafness, three of which had tinnitus aurium.

Three cases of otitis media, one of which had tinnitus aurium, and the other two tympanic membrane findings.

Eleven cases of general impaired hearing, nine of which had associated tinnitus aurium, one unilateral antrotomy, one lues.

Eleven cases of otosclerosis, eight of which had tinnitus aurium, and the others tympanic membrane findings.

Two cases of deafness associated with tinnitus aurium.

Among these cases there was no special selection made, but each was treated in its proper order. The patients invariably reported a refreshed feeling following the massage. Of the total number, only two (one otosclerosis and one impaired hearing) actually believed their hearing facilities to be improved. Audiometric tests, however, showed no evidence of improvement. One woman (otosclerosis), who was talented musically, reported that the treatment enabled her to have keener enjoyment in piano playing, since sounds were clearer and tones much richer. Accordingly, further tests were made on this patient, but the improvement was due merely to the efficacy of the massage, a point which had been

previously noted in many patients. None of the other patients reported any auditory improvements due to the hormone therapy. On the basis of these careful tests with Zajicek's hormone ointment, the authors came to the conclusion that it has absolutely no value.

Experimental work of definite value on prostigmin therapy of deafness was presented by Racine.[46] He stated that Davis and Rommel (*Arch. Otolaryngol.*, 1939; *Clin. Med.*, 1939) report "miraculous" results with prostigmin in the treatment of deafness. With interest but skepticism they undertook similar studies.

They used a Marconi-Ecko audiometer. The hearing curve was recorded before and 15 and 30 minutes after intravenous administration of 0.5 or 1 mg. prostigmin. The tests were carried out on 30 subjects, normal as well as deaf persons.

They were particularly interested in the effect of the drug on hypersympathicotonic deafness. As shown in the accompanying charts, regardless of the site of the lesion causing deafness, the modifications caused by prostigmin never exceeded 5.6 dcb. for frequency C_2, 4.7 dcb. for frequency C_3, and 4.8 dcb. for frequency C_4 — these three frequencies being in the hypersensitive zone. An average maximal increase of 5 dcb., even in the range of articulate speech, is not sufficient to cause functional modification of deafness.

Theoretically, dystonic deafness due to hypersympathicotonia would be favorably affected by vagotonic drug. Actually, however, this is not the case since this drug, even when given intravenously, causes no change in local vegetative equilibrium in the ear or elsewhere.

Our studies on 30 subjects did not show a single instance of improved hearing acuity following prostigmin therapy.

(There were nine charts showing auditory curves.)

GROUP 3. SURGICAL TREATMENT.

A further modification of the labyrinth fenestration operation for improvement of hearing in otosclerosis was described by Kend.[47] His technique included the performance of a Blackwell radical mastoidectomy with a modification of the flap formation, the removal of the head of the malleus, and

the fenestration of the external semicircular canal. The operation is performed through a postaural incision under general anesthesia. Kend believes his technique alters the normal process of osteogenesis, in that the periosteum is depressed during the period of healing between the new vibrating membrane and the margins of the fenestrum, thus tending to insure a patent fenestrum. His routine also attempts to alter the calcium content of the body by placing the patient on a vitamin D-free diet, high calcium intake, low in phosphorus, also using vitamin D depressing foods and bovine ovarian extract for two weeks preoperatively and continuing for four weeks postoperatively.

In an article which strives to further advance his technique of the labyrinth fenestration operation, Lempert[48] repeats much of the information, ideas, etc., which he has presented in previous papers, particularly on the indications of the operation. He reports three surgical accidents. In one case, fracture of the vertical mastoid portion of the Fallopian canal occurred, causing a temporary facial paralysis, and in the other two cases the lateral sinus was punctured, but the bleeding was easily controlled. He also reports that two cases developed a serious labyrinthitis postoperatively, resulting in complete loss of the vestibular and cochlear functions. Two of his patients died of coronary thrombosis.

Lempert further reports on 39 revisions. In 21 cases fibrosis was found to be the cause of closure of the fenestrum. In six other cases osteogenesis was found to have decreased the size of the fenestra without resulting in complete closure.

In his summary of 150 cases which had received the fenestration operation, Lempert states that a permanent restoration of practical physiological hearing resulted in 93 cases, a marked improvement occurred in 11 cases, and a further impairment of hearing resulted in 14 cases, and the hearing remained unimproved in 32 cases.

In his theorizing on the cause of the hearing improvement following fenestration, Lempert has concluded that the improvement is not the result of decompression of intralabyrinthine fluid pressure, but rather the result of the mobilization of the perilymph and endolymph by air-borne sound. He describes in detail how the fenestra should be made and how it should be covered in order to obtain permanent results.

He experimented with the use of Thiersch grafts placed on the tympanomeatal membrane before applying it over the fenestrum and claimed improved results. He concludes the article by emphasizing several ways in which the fenestration operation is superior to the use of a hearing aid.

Campbell[49] presented a detailed report of 12 cases on whom the labyrinth fenestration operation for deafness had been performed. This article was in many respects supplementary to a previous report, in that it recorded the progress of the eight cases which had been reported previously. The follow-up report on these first eight cases was recorded. Three of them retained improvement as follows: one an average gain for conversational frequencies of 31.7 dcb. for two and one-half years after operation; another an average improvement of 16.6 dcb. for two years after operation; and the third, an average improvement of 26 dcb. for 14 months after operation. The remaining five cases of this group were unsuccessful. A summary of his results in the 12 additional cases follows:

1. Six patients received improvement of hearing, and six patients lost some hearing as the end-result.

2. This generally poor result was attributed largely to improper selection of patients in the majority of cases.

3. Five of these 12 cases possessed the proper operative indications, four of whom retained improved hearing.

4. Of the seven patients who did not possess the proper operative indications, two retained improvement of hearing.

5. Five patients showed evidence in the tympanic membranes of having had suppurative otitis media at some previous time. Two of this group retained improvement of hearing.

6. In five of the 12 operated cases the malleus head was not removed. One of this group retained a slight improvement of hearing.

7. The results indicated that at least some of the cases in which there had been middle ear abscesses at some previous time could be improved by the operation.

Ersner and Myers[50] recorded their results in 17 cases of deafness in which the labyrinth fenestration operation was

performed. They stated that 38 operations had been performed since 1938, of which four patients were restored to practical physiologic hearing, having been observed for periods varying from six months to two and one-half years. Of the remaining 34 cases, all the fenestra made closed after periods varying from several weeks to eight months. All cases were done according to Lempert's endaural technique. The authors emphasized the importance of a careful history and medical studies preliminary to operation. Only 17 of their cases were reported in detail with audiograms. They discussed some interesting features of the postoperative reactions in their patients, such as incidence and extent of the vertigo, the state of the hearing, the fistula reaction, and wound infection and healing. They were under the impression that the tendency for the fistula to close was a factor over which at present there was no control.

Under the title of "Modern Treatment of Otosclerosis," Nasiell[51] has speculated somewhat on the cause and diagnosis of otosclerosis and reviewed incompletely the early progress made in the treatment of this condition by fenestration of the labyrinth. He considered that attempts thus far had met with failure but felt that the solution of the problem was close at hand. He mentioned that new difficulties had arisen as to the differential diagnosis between otosclerosis and adhesive processes in the middle ear. He considered, however, that permanent artificial sound conduction to the labyrinth could be just as effective for fixed stapes in the adhesive process as for the same component in otosclerosis. Nasiell further stated that the results of his tests of the method by which Max Meyer was recently reported to have obtained splendid improvement of hearing in patients with otosclerosis, *i.e.*, the suboccipital withdrawal of a considerable quantity of cerebrospinal fluid, had been negative.

Lempert,[52] in further elaboration and modification of his endaural technique for fenestration of the labyrinth in otosclerosis, describes in detail his operation to produce the "Fenestra Nov-Ovalis." He was led to seek an improvement of his former technique because of the postoperative closure of the fistulas in 88 of 300 cases in which the external semicircular canal was fenestrated.

An investigation of the closed fenestrae by reoperation of 72 of the 88 cases revealed that some of them had closed by

bony regeneration and some had closed or partly closed by formation of fibrous connective tissue. The chief reasons for the bony and fibrous tissue formation closing the fistulas appeared to be the inability of the small perilymph space of the external semicircular canal to absorb the blood accumulating within it, and also the fact that Shrapnell's membrane could not be stretched sufficiently to cover the fenestrum.

The technique of this new procedure is the same as that previously described by Lempert, except that the incus is removed and the fenestrum made in the vestibule instead of the external semicircular canal. With the fenestrum in this location it is possible to place Shrapnell's membrane itself over the opening. In a series of cases, Lempert is placing an obturator frame, made of platinum and iridium, into the vestibular fenestrum before covering it with the tympano-meatal membrane. He has created the fenestra nov-ovalis in 75 patients in the past 18 months and claims that in not one of these has the fenestrum closed. This has led him to abandon fenestration of the external semicircular canal.

Passe[53] attempted treatment of otosclerosis by cerebrospinal puncture. He stated that the effect upon otosclerosis of simple lumbar puncture has been tried many times without result. A patient who had a head injury and was being investigated by lumbar tap and air replacement for ventriculography stated three hours after this procedure that he no longer had the head noises and his hearing was improved — he had had deafness for four and one-half years. Deafness appeared to be due to otosclerosis — complete with family history.

On the following day the hearing remained improved but tinnitus returned, but was of different character.

A month later the condition remained the same as the day following the spinal tap. He agreed to have the procedure repeated. Eighty cc. of cerebrospinal fluid were withdrawn and 60 cc. of air replaced. Fluid was normal in pressure and contents. Tinnitus was relieved for 25 hours and hearing further improved. Four months later, tinnitus persisted but hearing continued improved.

Six cases were then tried. All patients experienced considerable degree of improvement in hearing and temporary subsidence of tinnitus was present.

The patient is fit to return to work on the third or fourth day.

No audiometric records were made — spoken voice was the test.

Campbell[54] presented a report and an evaluation of his results in a series of 30 patients on whom he had performed the labyrinth fenestration operation for chronic conductive deafness. These cases had been operated for periods longer than eight months. An analysis of some of his cases had shown that after an average time of three and four-tenths months following operation there was practically no further loss of hearing. He concluded from this that the end-result had been reached in these cases as far as the reparative process following the operation was concerned.

A summary of his results in these 30 cases was as follows:

1. Twenty-one had suitable operative indications.

2. Of these 21, 13 received an improvement of more than 14 dcb. each at what was considered the end-result.

3. These 13 patients received an average of 21.8 dcb. improvement of the hearing.

4. The eight unsuccessful cases of the 21 with suitable operative indications received an average loss of 4.8 dcb. over the preoperative level.

5. Of those 17 cases which had thickened, retracted, scarred and perforated tympanic membrane, 10 received improvement of hearing.

6. Those cases which were revised were not successful, two receiving no improvement, one losing 10 dcb., and the fourth gaining only 8.3 dcb.

7. The tinnitus paralleled the change in hearing in all cases.

8. Those nine cases which had unsuitable operative indications received no improvement of hearing as the end-result.

In his conclusions, Campbell stated that in the great majority of successful cases the end-result would not be the best hearing obtained a short time after operation but would be somewhat reduced from that highpoint, but would be suffi-

cient to be of practical importance providing the preoperative deafness had not been of too great a degree. He also felt that the postauricular approach gave several important advantages over the endaural route. He advanced the theory that the retention of the open fistula depended on the union of the opposing soft tissue surfaces at the site of the fistula before bone regeneration had proceeded to the point of closing the fistula. Such interposing of soft tissue between the bony edges should prevent the spread of bone over the fistula.

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2035 Delancey Street.

NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLARYNGOLOGY.

Meeting of Nov. 19, 1941.

Effect of Tonsillectomy on Respiratory Infections in Children. Dr. Albert D. Kaiser (by invitation).

Respiratory infections in infants and in children comprise the most common group of illnesses for that age group. Statistical evidence points to a lowered mortality and morbidity rate from these diseases during the last 10 years, but no specific measures have been developed that assure reasonably certain control of these common infections. Though the common respiratory infections termed colds, sore throat or tonsillitis, laryngitis, bronchitis and pneumonia represent the usual manifestations in infants and in children, such complaints as otitis media, cervical adenitis and sinusitis must be considered as a part of or as sequelae of respiratory infections.

Children at all ages may contract any of the respiratory infections but they are all most prevalent in the age grouping of 3 to 7 years. It is also certain that geographical location and climatic advantages influence the occurrence of these complaints.

Definite progress has been made in the control of respiratory infections. Advances in the science of nutrition, particularly with reference to vitamin and mineral needs, have played a part in developing resistance to respiratory infections. To immunological studies and recently to chemotherapy can be ascribed significant gains in the management of these common infections.

For at least two decades there has been a strong conviction among the medical profession and in lay groups that the tonsils and adenoids play a significant part in predisposing to or prolonging respiratory infections in children. It is therefore important to study the role of the tonsils and adenoids.

A group of 4,400 children was followed for a period of 10 years, beginning when they were between the ages of 4 and 7 years and continuing into their high school period. At the time of their initial examination the indications for tonsillectomy were numerous but, in general, about the same for the entire group. For various reasons only one-half of the total submitted to operation, so the children unoperated upon served as controls.

Conclusions: The tonsils are undoubtedly responsible for many upper respiratory infections, especially in children subject to tonsillitis and to cervical adenitis, and can be considered as playing an important role in these infections. Tonsillectomy is desirable in such children.

The adenoids appear to be a causative factor in some children who develop frequent colds and middle ear infections. In the age period of 3 to 7 years the adenoid structures are particularly a menace to many children and their removal does assure some protection against similar recurrent attacks. Adenoidectomy is advised in such children, either alone or together with tonsillectomy.

For the relief of and protection against sinusitis and nasal allergy, no statistical evidence is available in favor of the removal of the tonsils or adenoids, or both. The tonsils and adenoids seem to play no significant part in the production of these clinical manifestations.

Evidence obtained in this study does not support the opinion that laryngitis, bronchitis and pneumonia can be reduced in incidence by removing the

tonsils and adenoids. It appears that laryngeal, bronchial and pulmonary infections are not dependent upon the presence or absence of the tonsils, and that these lymphoid structures do not play a significant role in lower respiratory diseases.

Improved hygienic and dietary measures, together with specific therapy of pulmonary diseases, have brought about a condition which justifies the statement that the role of the tonsils and adenoids in the average child is not as important in the control of respiratory infections today as was thought to be the case a decade or two ago.

Relationship of Upper Respiratory Infections to Chronic Arthritis.

Dr. Ralph Henderson Boots and Dr. Robert L. McCollom.

(1) Routine tonsillectomy is not indicated in any form of arthritis.

(2) A diagnosis of the type of arthritis should be made before considering any relationship to a respiratory or any other focus.

(3) Certain types of arthritis, such as osteoarthritis, gonorrheal arthritis, tuberculous arthritis, etc., are definitely *not* related to upper respiratory infection.

(4) In rheumatoid arthritis particularly, it is wise to remove tonsils which seem diseased and to treat sinus infections, not because of any proven etiological relationship but because anything which improves the health of these patients seems to help their arthritis.

(5) The question of tonsillectomy or treatment of sinusitis in any given case still is largely one of clinical judgment of the physician. Decision regarding such treatment is better made by combined study by the internist and otolaryngologist.

(6) The necessity of taking a very careful history of upper respiratory infections in these cases is stressed.

(7) Early treatment of the sinuses and early removal of the tonsils is necessary if one is to expect any benefit in the rheumatoid arthritis group.

(8) The results in private practice are probably better than in the clinics as they are usually seen earlier and possible foci are treated and eradicated at an earlier stage of the disease.

Tonsillectomy and Acute Nephritis. Dr. John D. Lyttle.

No doubt exists as to the close relationship between the pathogenesis of acute nephritis and hemolytic streptococcus infection, chiefly of the upper respiratory tract. Clinical, bacteriological and immunological studies demonstrate an invasion of streptococcus hemolyticus in over 90 per cent of the cases of acute glomerulonephritis. The infecting organism may enter through any portal, but in the great majority of cases it is via the upper respiratory tract and associated structures — exactly which ones matters little for our purposes since these structures are continuous as well as contiguous.

The type of infection encountered with acute nephritis is quite different from that with rheumatic fever. Instead of a mild, transient sore throat, such as precedes the rheumatic attack, the child who develops nephritis has a deep infection, such as mastoiditis, cervical adenitis or peritonsillar abscess. The uncomplicated case of tonsillitis or scarlet fever does not develop nephritis. This is often discovered in the course of routine examination of a child who has been brought to the hospital or clinic because of mastoiditis or cervical adenitis.

Admitting a relationship between hemolytic streptococcus invasion of the upper respiratory tract and acute nephritis, the question arises as to what we can do about it and, in particular, as to what is the effect of tonsillectomy.

There is no evidence that tonsillectomy plays an important role in preventing acute nephritis. Children who are destined to develop acute nephritis are subject to repeated attacks of upper respiratory infection, and tonsillectomy does not seem to lessen materially the incidence of such attacks in this group. Possible prophylaxis against nephritis might be achieved through the management of the preceding infection. If this primary infection could be treated so as to prevent a recrudescence in the second to third week, it is conceivable that some cases of nephritis might be prevented. Obviously, tonsillectomy and adenoidectomy do not prevent the septic complications which accompany acute glomerulonephritis. All forms of treatment in scarlet fever are said to lessen complications but the data is not convincing. Chemotherapy offers an opportunity for a carefuly controlled study of this question.

Should tonsillectomy be advised in cases of acute nephritis and, if so, when should it be performed? The object of tonsillectomy in these cases is presumably to prevent the development of chronic nephritis by removing a focus of infection which may be causing persistent renal damage, and to lessen the incidence of upper respiratory infection which might also damage the kidney further. There is no basis in fact for this conclusion. If we could predict which children were going to develop chronic nephritis, that question might be answered.

It is true that in the majority of cases the activity of the nephritis parallels that of the infectious process, and that urinary changes of definite degree persist and recur with persistence or recurrence of the infection. But I have seen prompt healing of nephritis in the presence of persistent infection, and I have also seen nephritis progress to the subacute and chronic stage without any clinical evidence of infection. These events can all take place regardless of the presence or absence of tonsils, or of tonsillectomy in the acute stage.

Once chronic nephritis has become established, I have never been convinced that any attack on foci of infection has altered the course of the disease. Our experience is in accord with that in Baltimore — that tonsillectomy did not prevent infection or exacerbation of the nephritis, and that other factors must operate for recovery or progression.

In the face of divergent opinions and without any rational basis of procedure, what is a reasonable attitude for the clinician to take? While we are ignorant of the pathogenesis of glomerulonephritis, there does appear to be in some individuals a relationship between upper respiratory infection and nephritis, and if any of the generally accepted indications for tonsillectomy are present in a case of either acute or chronic nephritis, the operation should be performed. Nephritis itself is not to be considered an indication for tonsillectomy and adenoidectomy.

Tonsillectomy is an elective operation, and the proper time to perform it can best be decided for each individual case, preferably when the signs and symptoms of the nephritis have subsided, and when the pharynx is not acutely inflamed. When this is done, severe reactions have not been encountered in my experience, but I must admit that the beneficial effects are not proven.

DISCUSSION.

DR. WESTLEY M. HUNT: Someone sitting near me during the reading of these papers remarked: "Our mastoids have gone, and here go our tonsils!"

I do not think that is quite true of the papers presented tonight. I hope none of the gentlemen were influenced to be too kind because they were appearing before a group of otolaryngologists. I have been greatly instructed this evening. These papers are hard to discuss because they have brought us an analysis of things about which we knew very little a short time ago. I feel a bit as Dr. Lyttle did, that perhaps most of my discussion will be talk.

Dr. Kaiser in his paper has brought out the very interesting fact that tonsillectomy does not particularly minimize the lung infections of bronchitis

and pneumonia. In the upper respiratory infections he feels the procedure is definitely beneficial.

Dr. Boots' paper is going to be discussed by someone connected with his clinic, so I shall not go into his findings. I am not yet able to estimate whether tonsillectomy will help in a given case. Dr. Boots gave us indications which will allow us to appreciate the reason for the definite results we have had in some cases, and the others we have operated upon in which we have not attained satisfactory results.

Dr. Lyttle's paper on the findings in nephritis is really a medical paper and is again instructive to me and, I am sure, to most laryngologists. This whole discussion tonight emphasizes the importance of closer co-operation between the different departments of our hospitals. I have realized from these presentations the opportunities we have missed in the matter of collaboration.

I have no tabulations which could either support or refute the findings of the speakers tonight. These are very interesting and valuable papers, and I wish I were better qualified to discuss them.

Dr. Robert L. McCollom (by invitation): I should like to discuss Dr. Lyttle and Dr. Kaiser's papers only very briefly, because I do not feel qualified to speak on their topics. I have not had enough experience in following cases in their fields.

Dr. Kaiser mentioned allergic diseases and said that infection did not enter into that picture very much. I should like to take one exception — cases of childhood asthma. We frequently see attacks of asthma initiated by a cold or upper respiratory infection; it may not be the basic factor but it does seem to initiate the attack, and it causes many of the attacks that we see. If we can cut down the incidence of upper respiratory infections, possibly by treating the sinuses or tonsils, there may be fewer attacks of asthma.

Dr. Lyttle, in dicussing nephritis, mentioned cervical adenitis and also peritonsillar abscess. I have a feeling, without any figures to back it up, that if the tonsils were not there, the patient would be less likely to develop peritonsillar abscess and, as a consequence, might not have nephritis as a complication.

In discussing Dr. Boots' very thorough paper, I should like to stress, aside from trying to differentiate the type of arthritis involved, the necessity of taking a very careful history with regard to upper respiratory infection. In the cases we have followed at the Medical Centre, at least 50 per cent have had a very definite history of upper respiratory infection. As Dr. Boots said, 20 per cent of the cases in the rheumatoid arthritis group seem to have the onset of arthritis occur about a week or two weeks after the upper respiratory infection — the same period as we find in nephritis following sore throat or peritonsillar abscess, or a cold or grippe. I do feel that in that particular group where the arthritis follows such an episode, treatment such as removal of the tonsils is apt to be effective and that we do get some improvement in the arthritis following operation. The percentage of cases that are helped is not great, it is true, but some are definitely benefited and I do not think we should neglect to remove the tonsils in cases with such a history.

I should like to stress early treatment. The cases coming to the Medical Centre in a great many instances are not seen early; some have had arthritis for some time, some have been seen elsewhere before getting to the clinic. If any benefit is to follow removal of the tonsils for arthritis, the operation must be performed early in the disease, otherwise it is a case of shutting the barn door after the horse is stolen.

Dr. Boots covered the different types of arthritis pretty thoroughly. I have discovered in following about 180 cases that about 15 per cent of the rheumatoid group seem to be helped following removal of the tonsils. Even though that is not a large percentage, anyone who has had arthritis will feel that it is worth while if any at all are helped. Another group, not so large,

we call the chronic rheumatic fever group, with a high antistreptolysin titer. I have not followed many of those cases but most of them had a history of upper respiratory infection which seemed to initiate the joint pains. The results of tonsillectomy in that group seem to be very encouraging, but I haven't followed enough of them to give conclusive figures.

The focal infection group mentioned by Dr. Boots is very rare and is more likely to be associated with acute antral infection than with acute tonsillitis. If they have an acute antrum and it is treated, it usually clears up.

There is a very large group which Dr. Boots classed as coming under fibrositis or myositis cases. They are really not arthritis cases; but they do have muscle pains and pains around the joints, and they are quite often called arthritis. That group is most commonly associated with upper respiratory infections. I believe we see a lot of them in private practice, and I think they respond to any method of treatment we may use. If they have a sinusitis and we clear it up, the fibrositis will usually clear up. Another thing is usually true of these cases: in private practice the results are probably better than we would see in this group in the clinic. In private cases the patients are usually seen earlier; they usually get pretty careful co-operation between the nose and throat man and the internist, and the results are a little better.

I do not believe we can do much for the osteoarthritis group. I have seen an occasional case that has had a tonsillitis followed by operation, and it seemed for a while that the osteoarthritis might be better, but in the long run I don't think that that particular group responds very much to any ear, nose or throat treatment.

Dr. Miles Atkinson (by invitation): On this very vexed question of focal infection and its relation to chronic and rheumatoid arthritis: I happened to practice at one time in an English spa, Bath, where this was a constant trial to my colleagues and to myself. The question was constantly coming up as to whether tonsils should be removed in cases of rheumatoid arthritis — and I am not talking of osteoarthritis, nor of frank focal arthritis which offers no particular diagnostic difficulty and requires no great clinical acumen — but of true rheumatoid arthritis of which we saw pictures on the screen this evening. There were in fact two schools of thought in Bath, where a great many cases of rheumatoid arthritis are seen, as to what was the pathology of this condition; and, after all, it is on the pathology of rheumatoid arthritis that our treatment, as far as the nose and throat and focal infection are concerned, must rest. Those two schools of thought were: (1) rheumatoid arthritis is an infective arthritis — not necessarily from such a focus of infection as teeth, tonsils or sinuses, but, nevertheless, an infective arthritis. (2) The other school held that it is nothing of the sort, but that it is a metabolic, or probably metabolic, disturbance — at any rate, not infective. There is much evidence that it is disease sui generis, probably metabolic, and, if so, then one could not expect to make very much impression upon it by removing a more or less hypothetical focus of infection; in other words, by scattering tonsils right and left, draining sinuses not needing drainage, etc. I must confess that my experience has inclined me very strongly toward that second school. I can't give any figures for it, I'm afraid, but I can report it as a general experience, that I have laid many a tonsil on the altar of the great god, focal infection, but very, very seldom has he smiled.

Dr. Albert D. Kaiser: In reply to Dr. McCollom's remarks, I am fully aware of the fact that attacks of asthma may be due to infection, but I think I did state in my paper that I was talking about allergic rhinitis and not asthma. I think the allergists with whom I have conferred quite agree that it rarely happens that an allergic rhinitis is benefited by tonsillectomy. Rheumatic disease in children is quite different from that in adults. My own experience, both with nephritis and with rheumatic disease, has been similar to that of Dr. Lyttle and Dr. Boots. I do think we must weigh the desirability of a tonsil operation, and the question as to whether or not it is going to do any harm, and if we think it is not we ought to offer its possible benefits, particularly in rheumatic disease, until we have a better method of treat-

ment. Over a period of 10 years I have followed 1,200 children with rheumatic fever, and we have no evidence that tonsillectomy prevented rheumatic disease, or recurrence of rheumatic disease, but we do find that the mortality from rheumatic carditis over the 10-year period was decidedly less in children who had had their tonsils removed. It may be because we did not have repeated throat infections, or insults from repeated infections, for the mortality was 13 per cent in children in whom the tonsils were not taken out, and only 7 per cent in children in whom the tonsils were removed either in the period before they developed rheumatic disease, or during the period in which they had it. So, until we have some satisfactory drug or other means of treating rheumatic disease in children, I believe operation is still justifiable. Since we carefully study the child with nephritis before operation, I have not seen the ill effects following tonsillectomy, spoken of by Illingworth, of England.

As I heard at dinner, the use of the sulfonamide derivatives may affect the incidence of mastoiditis, and I am inclined to think it will. Not only will the powerful drug prevent septic complications but it may also turn out to be a partial preventive of rheumatic disease. But I think tonsillectomy will continue until we have further evidence that it is not necessary. The thing I condemn, however, is not the use of operation in these focal infections but indiscriminate operation without first giving the child the benefit of study; and I am glad to say it is not being done as freely or as needlessly as it was 10 or 15 years ago. I think consideration is now being given to other possible causes of trouble.

Dr. John D. Lyttle: Dr. Moyle asks whether we can prevent nephritis by keeping a child in bed for a longer period after the initial infection. I do not think we have any way of proving that any measures taken in convalescence prevent nephritis. The great majority of children with streptococcus infections are allowed up from a few days to a week after the fever subsides. The great majority of children do not develop nephritis under these circumstances. No rule can be made; each case must be decided on the clinical findings. It has always seemed to me that the child with streptococcus disease and a rash gets better treatment than the child without a rash.

As to the question about focal nephritis, I would say that I am unable to make an anatomical diagnosis in these conditions and my attitude would be the same in all forms of acute glomerulonephritis.

Dr. Page Northington, Chairman,
20 East 53rd Street, New York.

Dr. Ward C. Denison, Secretary,
20 East 53rd Street, New York.

BOOK REVIEW

Endotracheal Anesthesia. By Noel A. Gillespie, M.D., B.Ch., M.A. (Oxon); (R.C.S., Eng.); Research Associate and Resident in Anesthesia, University of Wisconsin, State of Wisconsin General Hospital; Former Senior Resident Anesthetist, The London Hospital; Hospital Assistant Instructor in Anesthetics, London Hospital Medical College and Dental School, etc. One hundred eighty-seven pages with Bibliography and Index. One color plate and 37 illustrations. Madison, Wis., The University of Wisconsin Press. 1941. Price $4.00.

From its introductory justification of the dual use of the words "endotracheal" and "intratracheal" to identify identical procedures, to its final chapter relating tracheal intubation to resuscitative therapy, Gillespie's "Endotracheal Anesthesia" interestingly presents a wide variety of applications of that technique that cannot fail to profit the anesthetist who digests the essence of that monograph and the significant related literature which it recapitulates.

After historically tracing the antiquity of endotracheal experimentation and its consequent development of precursors of present techniques, the author clarifies widely varied applications of that method of administration of modern anesthesias.

His detailed treatment of the intubation apparatus now used and his recommendations of practical procedures for manipulating them constitute a distinct and valuable contribution to the literature relating to this modern aspect of anesthetic administration. H. L.